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MENINGO-ENCEPHALITIS DUE TO COXSACKIE B VIRUS IN SOUTHERN RHODESIA

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In the summer of 1953-54 several cases of meningo-encephalitis were admitted to the City Hospital, Salisbury. Some were sent to hospital with a provisional diagnosis of poliomyelitis, others were suspected to be suffering from involvement of the central nervous system complicating glandular fever. While the cases were being received in the isolation hospital, a minor epidemic of Bornholm's disease was running its course in Salisbury but, because this condition is not notifiable, knowledge of its existence did not reach us until some time later. Virus studies were carried out in the laboratories of the Poliomyelitis Research Foundation in Johannesburg, and from a significant number of cases a virus of the Coxsackie B-group was isolated. This paper describes the clinical and laboratory findings in a representative sample of these cases.

CLINICAL FINDINGS

The cases described were all admitted to the City Hospital, Salisbury. The case histories are summarized below.

Case 1

R.C.O., a boy aged 6½ years, presented on 15 November as not sleeping, with large amounts of pus on posterior pharyngeal wall. Next day his temperature was 100·4° F. He was given sulphatriad, 0·5 g. 6-hourly. On 20 November he developed intense headache and began vomiting. He was now given aureomycin, 500 mg. 4-hourly. Next day he was admitted to hospital for observation as a suspected case of meningitis.

On admission, there was slight neck rigidity and slight stiffness of back, but no evidence of muscle weakness. Reflexes were present but the right knee-jerk and ankle-jerk were difficult to elicit. It was noted that he was a mouth breather and this was thought to account for the injected pharynx. Nothing abnormal was detected in the urine or the cerebrospinal fluid, which was clear and not under pressure and was reported on by the laboratory as follows: Protein 30 mg., sugar 70 mg., chlorides 700 mg., globulin negative. Cells: Total 410 per cmm.; polymorphs 51%, lymphocytes 48%, monocytes 1%. No organisms were found on direct examination of smear, or after culture.

Progress: The temperature was of a remittent type for some days after admission. The neck rigidity had cleared up by 25 November, but slight spasm of the lumbar region on attempting extreme flexion persisted for another 2 weeks.

Treatment: The patient was given hot packs and radiant heat for his back and limbs. Aureomycin was continued until 26 November when the pyrexia disappeared.

A stool specimen sent to the laboratories of the Poliomyelitis Research Foundation in Johannesburg on 30 November yielded a virus of the Coxsackie B-group.

Case 2

M.J., a boy aged 6 years, was admitted to hospital on 24 November with a history of backache, headache, abdominal pain and vomiting for 3 days and suspected to be a case of poliomyelitis. On admission, temperature was 101° F, flushed face, sore throat, and some rigidity of the back on flexion. Reflexes were normal. Kernig's sign was negative and there was no weakness. The boy was constipated.

The cerebrospinal fluid withdrawn by lumbar puncture was clear and under slight pressure; cell count: leucocytes 1 per c.mm., red cells 1 per c.mm., chlorides 740 mg., protein 20 mg. No organisms seen in the direct smear.

The throat swab culture grew an occasional colony of haemolytic streptococci, but a later swab gave a negative culture for this.

Blood count: Total leucocytes 5,700 per c.mm.; neutrophils 39%, lymphocytes 53%, monocytes 4%, eosinophils 4%. The blood film for malarial parasites was negative.

Urine: nothing abnormal detected.

Treatment: Sulphadiazine was given for the throat condition, cascara and agarol for the constipation. Radiant heat and hot packs were used to relieve the back stiffness.

Progress: The temperature settled within 4 days of admission. The back spasm and the throat condition had disappeared by 28 November, but the child was kept under observation as a suspected case of non-paralytic poliomyelitis until 19 December, when he was discharged.

A stool specimen was sent for virus studies and a Coxsackie B-group virus was isolated.

Case 3

K.I.W., a boy aged 3½ years, was admitted to hospital on 28 November as a suspected case of meningitis with a history of headache, loss of appetite and backache for 4 or 5 days. About a week before admission he had an illness with high temperature for which no definite diagnosis was made.

On admission temperature was 103° F, neck rigidity and neck pain was present and there was slight pain in the lumbar region on flexion. The knee and ankle jerks were exaggerated and there was some ankle clonus; plantar reflexes were flexor and the lower abdominal reflexes were doubtful. Kernig's and Brudzinski's signs were negative. The cervical glands were enlarged, and there was slight injection of the pharynx.

The cerebrospinal fluid withdrawn was opalescent but not under pressure; cell count: leucocytes 704 per c.mm., erythrocytes 2,069 per c.mm., chlorides 740 mg., protein 40 mg. Microscopical examination of a direct smear showed numerous mononuclear leucocytes, but no organisms.

Blood count on 3 December showed: Total leucocytes 7,800; neutrophils 54%, lymphocytes 40%, monocytes 6%. Repeated on 9 December, the picture was: Total leucocytes 17,000; neutrophils 58%, lymphocytes 38%, monocytes 4%.

Paul-Bunnell tests on 2 occasions gave titres of 1 : 14 and 1 : 7 respectively.

Treatment: This child was given 400,000 units of crystacillin *stat.* and 200,000 units of procaine penicillin b.d. with sulphadiazine, 1·5 g. *stat.* et 0·5 g. 4-hourly. As the symptoms persisted this treatment was discontinued on the 4th day and aureomycin spercoids, 1 dr. 4-hourly, substituted. Symptoms had cleared by the 10th day, when all drugs were discontinued. The child was detained in hospital for a further 11 days.

A Coxsackie B-group virus was isolated from a specimen of stool collected on 4 December 1953.

Case 4

R.D.H., a young woman aged 25 years, was admitted to hospital on 6 January 1954 as a suspected case of meningo-encephalitis with a history of sore throat and lassitude for 13 days. For the past week she had suffered generalized muscle pains, and 2 days before admission she developed intense headache with painful stiffness of the neck.

On examination, the patient had a furred tongue, painful cervical glands, a palpable spleen, and an irregular cardiac rhythm with a systolic bruit at the apex. She obtained relief from the symptoms after the lumbar puncture and then made a steady recovery.

The cerebrospinal fluid showed: leucocytes 20, erythrocytes 1, chlorides 780 mg., proteins 25 mg. The film showed mononuclear cells, but no organisms.

The blood count on 4 January showed: Neutrophils 73%, lymphocytes 25%, monocytes 1% and eosinophils 1%. Repeated on 8 January, the picture was: Total leucocytes 9,900; neutrophils 55%, lymphocytes 36%, monocytes 1% and eosinophils 8%.

A Coxsackie group-B virus was isolated from a specimen of faeces sent for virus studies.

Case 5

W.J.O.H., a young man aged 28 years and a brother of the preceding patient R.D.H., was admitted to hospital on 8 January 1954 as a suspected case of meningo-encephalitis, a relapse following an attack of Bornholm's disease of 7 days' duration. He had experienced acute gripping pain over the right side of his chest, pains in the back, listlessness and general malaise for a week previously. Then 2 days before admission he developed pain in his head and neck aggravated on coughing.

On examination, temperature was 102° F, inflamed pharynx, furred tongue, but no enlarged cervical glands and the spleen was not palpable. Reflexes were present and slightly exaggerated except for the plantar reflexes which could not be elicited. Neck showed some rigidity on flexion.

The cerebrospinal fluid on 9 January showed: leucocytes 19 per c.mm., erythrocytes 1 per c.mm., chlorides 780 mg., protein 30 mg.

Blood count on 9 January showed: Total leucocytes 13,400; neutrophils 63%, lymphocytes 33%, monocytes 4%.

Progress: On 11 January he developed headache, which was aggravated by sitting up. Later he complained of muscle pains, especially over the left shoulder. After discharge from hospital on 19 January he developed a further attack of muscle cramp and pains.

A Coxsackie B-group virus was isolated from a specimen of faeces sent for virus studies.

LABORATORY STUDIES

Stool specimens collected in bottles containing 30-50% glycerin-saline were sent by air from Salisbury to Johannesburg. Many, but not all, were received in the laboratories on the day of despatch.

1. TECHNIQUE

These specimens were then prepared for inoculation into baby mice and tissue-culture tubes in order to attempt to isolate Coxsackie virus or poliovirus.

A 10% emulsion of faeces was prepared in physiological saline. To this a quarter volume of anaesthetic ether was added and allowed to act in a refrigerator (4° C) overnight. The suspension was then centrifuged at 3,000 revolutions for 1 hour and the clear aqueous phase withdrawn and transferred to a 25-c.c. McCartney screw-capped bottle. This was placed in an incubator at 37° C for 2 hours to evaporate most of the dissolved ether. Penicillin, 100 units per c.c., and streptomycin, 100 μg. per c.c., were added for their bacteriostatic effects. A litter of 7 one-day-old mice were then inoculated subcutaneously with 0·05-c.c. amounts of the suspension.

This litter was observed for 14 days for signs of illness, especially for tremors, weakness and paralysis, and for whitening of the interscapular fat pad.

When a mouse developed any of these signs or even a suspicion of them it was sacrificed: the viscera were removed and portions of the brain, heart, lungs, liver, spleen, pancreas, kidneys, suprarenal, and limb muscles were placed in Bouin's solution to fix, prior to the preparation of histological sections. These were examined microscopically for specific lesions such as characterize the Coxsackie virus infections in baby mice.

The remainder of the carcase was then minced and a suspension prepared for passage into another litter of 7 one-day-old mice. Often when the first litter showed indefinite signs and histological lesions, the mice of the first or later passages showed definite signs and typical lesions.

The original suspension was also inoculated into tissue cultures of monkey testis in an endeavour to isolate poliovirus. The tissue cultures were observed for 14 days for signs of degeneration of the cells. If this was apparent, or even if there was suspicion of cytopathogenic effect, some of the fluid was withdrawn and passaged into a further series of tubes. If a virus was isolated it was identified by neutralization tests with the type antisera against the 3 types of poliovirus respectively.

2. RESULTS

In none of the 5 cases was poliovirus isolated from the specimen of faeces.

From each of the 5 a virus of the Coxsackie B-group was isolated. Details of the isolations in individual cases were as follows:

Case 1 (*R.C.O.*)

Dec.53
2 Suspension of faeces inoculated into a litter of baby mice.
7 1 mouse dead, 1 paralysed (arm), 1 with tremors, both kept for passage, 4 weak.
8 3 paralysed, 1 with tremors.
7 Mouse-carcase suspension passaged into litter of 7 baby mice.
10 2 weak.
11 1 dead, 2 showed tremors, 1 kept for passage.
12 3 dead, 1 weak, kept for passage.
7 Original suspension of faeces again inoculated into litter of 7 baby mice.
10 2 weak.
11 2 with tremors.
12 All 7 with tremors, 1 kept for pathology.

Histological sections showed early acute necrosis and acute inflammation of the fat pad, and several foci of acute necrosis and inflammation of the heart muscle; lesions characteristic of Coxsackie group-B virus infection.

Case 2 (*M.J.*)

Dec.53
2 Suspension of faeces inoculated into a litter of 7 baby mice.
7 4 dead, 2 weak, one kept for passage.
7 Suspension of mouse carcase passaged into another litter of 7.
10 All mice weak, 1 kept for passage.
11 4 dead, 2 paralysed, 1 kept for pathology.

Histological sections showed acute necrosis and inflammation of the fat pad, and foci of acute necrosis and inflammation in the heart muscle; lesions characteristic of Coxsackie group-B virus infection.

Case 3 (*K.I.W.*)

Dec.53
9 Suspension of faeces inoculated into a litter of 7 baby mice.
17 1 dead, 2 paralysed, also showing tremors, both kept for passage and pathology.

Histological section showed degeneration and disappearance of neurones and some softening of the white matter of the brain, with extensive necrosis, inflammation and calcification of the fat pad; lesions characteristic of Coxsackie group-B virus infection.

Case 4 (*R.D.H.*)

Jan.54
13 Suspension of faeces inoculated into a litter of 7 baby mice.
18 1 dead, 1 moribund, kept for passage, 1 showed tremors.
19 1 paralysed (arm), 1 showed tremors. 1 weak.
18 Mouse carcase suspension passaged into a litter of 7 baby mice.
21 All dead.
21 Mouse carcase suspension passaged again into a litter of 7 baby mice.
24 1 dead, 1 very weak and kept for passage.
25 3 dead, 2 paralysed, kept for passage, and 1 kept for pathology.

Histological section showed extensive early acute necrosis and inflammation of the fat pad, and foci of acute necrosis and inflammation in the heart muscle; lesions characteristic of Coxsackie group-B virus infection.

Case 5 (*W.J.O.H.*)

Jan.54
13 Suspension of faeces inoculated into a litter of baby mice.
17 1 mouse weak.
18 2 dead, 2 very weak, 1 kept for passage.
18 3 weak.
19 Mouse carcase suspension passaged into a litter of baby mice.
22 All 7 baby mice paralysed, 3 kept for passage and pathology.

Histological sections showed early acute inflammation of fat pad and small foci of acute necrosis and inflammation in heart muscle; lesions characteristic of Coxsackie group-B virus infection.

3. IDENTIFICATION AND TYPING OF THE ISOLATED VIRUSES

The viruses so isolated were typed in baby mouse protection tests against antisera prepared from the type strains of the 4 known types of Coxsackie B viruses. These type strains were kindly sent to us by Dr. G. Dalldorf.

In the tests 0·5 c.c. of neat serum was added to a suspension of mouse carcase containing approximately 1,000 MLDs of virus. The mixture was thoroughly shaken and incubated for 1 hour at 37° C, and then inoculated in 0·05 c.c. amounts subcutaneously into 5 of a litter of 7 or 8 baby mice. The remaining 2 or 3 of the litter (identified by having their tails docked) were inoculated with the virus suspension only.

The results of these baby mouse protection tests were as follows:

Antisera	*Virus Strains*				
Coxsackie	*R.C.O.*	*M.J.*	*K.I.W.*	*R.D.H.*	*W.J.O.H.*
B1 ..	0/5	0/5	0/5	0/5	0/5
B2 ..	0/5	0/5	5/5 (R5/5)	0/5	0/5
B3 ..	0/5	0/5	0/5	0/5	0/5
B4 ..	5/5 (R5/5)	4/5 (R5/5)	0/5 (R0/5)	5/5 (R5/5)	4/5 (R5/5)

Reading—Survivors/Total
(R=repeat test to confirm result)

The results indicate that the strains from 4 patients were Coxsackie group-B type-4 virus, and one a Coxsackie group-B type-2 virus.

COMMENT

This study has thus shown that each of these 5 cases admitted to hospital with the signs and symptoms of meningo-encephalitis—4 of whom showed a pleocytosis in the cerebrospinal fluid—were infected with a virus identified as a Coxsackie group-B virus. It is of interest to note that 4 were type-4 virus and one was type-2 virus.

As these viruses were isolated from the faeces and not from the cerebrospinal fluid, it cannot be stated definitely that they were the cause of the patient's illness. However, there are strong reasons for concluding that they were.

When these cases occurred, an epidemic of Bornholm's disease was in progress in Southern Rhodesia. From a number of patients a Coxsackie group-B type virus was isolated. One of the cases reported (No. 5, W.J.O.H.) had the symptoms of an attack of Bornholm's disease immediately preceding the onset of meningo-encephalitis, which developed as a complication of his illness. The other adult case and one of the children had symptoms of muscular pains suggestive of Bornholm's disease for several days before the onset of the signs of meningo-encephalitis. The other 2 children did not have typical signs of Bornholm's disease. This is in keeping with previous observations

noting that pleurodynia is more clearly defined in adults than in children, in whom abdominal symptoms and generalized muscular pains are more prominent.

In studies carried out in the Poliomyelitis Research Foundation Laboratories during the past year, Coxsackie group-B virus has been isolated from the cerebrospinal fluid and faeces of 6 patients, all of whom suffered from meningo-encephalitis, and 3 of whom had signs of Bornholm's disease immediately preceding the involvement of the central nervous system. These cases appear to be similar to those described here. Thus there is little doubt that in the cases in Salisbury the infection with Coxsackie group-B virus was responsible for the signs and symptoms of meningo-encephalitis as well as of Bornholm's disease.

It is now becoming clear that in addition to being the specific cause of Bornholm's disease, the Coxsackie group-B viruses commonly cause meningo-encephalitis, and indeed are one of the commonest causes of this condition in this part of the world.

It is also of interest to note that while this widespread epidemic of Coxsackie group-B virus infection was in progress, few cases of paralytic poliomyelitis were admitted to hospital. Similar observations have been made in Sweden and in the Union of South Africa. However, further investigation is necessary before it can be concluded that these 2 virus infections tend to be mutually exclusive—as the present evidence suggests.

SUMMARY

During the summer of 1953/54 a number of cases of meningo-encephalitis were admitted to the City Hospital, Salisbury, Southern Rhodesia. Some were sent to hospital with a provisional diagnosis of poliomyelitis, in others the involvement of the central nervous system was suspected to be a complication of glandular fever.

The findings in 5 of these cases are described. They were clinically characterized by intense headache, anorexia, nausea and vomiting, stiff neck and backache. One of the cases had the symptoms of an attack of Bornholm's disease, and 2 others muscular pains suggestive of Bornholm's disease, preceding the onset of meningo-encephalitis, which developed as a complication of their illness. Four of the 5 cases had a pleocytosis in the cerebrospinal fluid.

In studies carried out in the Laboratories of the Poliomyelitis Research Foundation, Johannesburg, a Coxsackie group-B virus was isolated from the faeces of each of these cases. In 4 the virus was identified as a Coxsackie group-B type-4 virus, and in the remaining case as a type-2 virus.

It is concluded that these infections with Coxsackie group-B viruses were responsible for the meningo-encephalitis as well as the myalgia suffered by these patients. It is noted that Coxsackie group-B virus is the commonest identified cause of 'benign' meningo-encephalitis in this region.

AN ORTHOPAEDIC CONGENITAL ABNORMALITY

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A European male, aged 48, consulted me complaining of a large bunion on the lateral side of each foot opposite the head of the 5th metatarsal. He had 6 toes on each foot; the radiographic appearance is shown in Fig. 1. This is the first case I have come across of a Y-shaped 5th metatarsal (bilateral). It was quite a problem to decide how to deal with the foot, there being no literature that I could lay my hands on; I eventually decided to remove the 6th toe only with its associated outer limb of the Y. The cosmetic and functional result has been most pleasing and satisfactory (see Fig. 2).

Fig. 1.

Fig. 2.

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VAN DIE REDAKSIE

VIRUSSIEKTES IN DIE ALGEMENE PRAKTYK

Soos bakteriese en protosoë-siektes onder meer volkome beheer gebring word, neem virusse in belangrikheid toe, beide op die gebied van navorsing en van die algemene praktyk. Onlangs het 'n Engelse geneesheer geskat dat ongeveer 'n kwart van sy besoeke aan pasiënte met virussiektes afgelê word.[1] Die probleem is des te ernstiger omdat daar tot dusver geen spesifieke anti-virusmiddels gevind is nie. Die sulfonamides en antibiotika is slegs van waarde met die bestryding van bakteriese komplikasies of as 'n terapeutiese filter met die bepaling van 'n diagnose. Sonder 'n spesifieke geneesmiddel is die algemene behandeling van 'n virussiekte van groot belang en die geneesheer kan heelwat tot die pasiënt se gemak bydra deur betyds raad te gee oor behandeling wat gewoonlik as onbelangrik beskou word. By twee van die 'vyf groot' virussiektes, nl. griep en kwaai masels [2] (die ander is pampoentjies, waterpokkies en rooihond) is die verpleging waarskynlik net so belangrik as in die geval van maagkoors. In beide gevalle mag 'n hoë koors dit wenslik maak dat die pasiënt elke halfuur met lou water afgespons word om stuiptrekkings te voorkom. Die beddegoed moet lig wees en die ventilasie in die siekekamer moet goed wees. Persoonlike higiene verdien ook aandag—dikwels is dit nodig om oumense in die hospitaal op te neem nie soseer nie om geneeskundige maar om maatskaplike redes. Dit is somtyds moeilik om kinders in die bed te hou of om ouers te oortuig dat dit nodig is as die redes daarvoor nie aangevoer word nie; dit mag wel die deurslag gee in die finale prognose van pampoentjies by seuns ná pubertiteit of in die geval van pasiënte wat besig is om van lewerontsteking te herstel. Dit is ook die moeite werd om aandag aan die dieet te gee, glukose vir die ketose van braking of hoë koors kan sekerlik goedkoper en makliker as 'n droë poeier gegee word as in een of ander patente vorm.

Die behandeling van simptome mag van die geneesheer diep kliniese kennis verg. Antibiotika het die skerpsinnigheid van menige 'n andersins skrander klinikus verstomp, want die wonderbaarlike eienskappe van hierdie middels het die lewe vir hom vergemaklik. Hy kan homself betreklik hulpeloos en verlore voel wanneer hy nie op hulle terapeutiese werking staat kan maak nie. Met virussiektes is beide verpleging en behandeling onontbeerlik. Warm drankies of 'n warm

EDITORIAL

VIRUS DISEASES IN GENERAL PRACTICE

As bacterial and protozoal diseases are brought under more complete control, the importance of the viruses looms larger both in the research laboratory and in general practice. Recently an English practitioner estimated that about one-quarter of his visits were made to see patients with viral illnesses,[1] and the magnitude of the problem is all the greater for the reason that no specific anti-viral curative remedy has yet been developed. The sulphonamides and antibiotics are of value in treatment only to combat bacterial complications or in diagnosis as a therapeutic filter.

In the absence of a specific remedy, the general management of a case of viral illness assumes greater importance, and the practitioner has the opportunity of adding considerably to the comfort of his patient by timely advice on a number of points usually considered to be trivialities. In two viral diseases, of the 'big five', viz. influenza and severe measles [2] (the others are mumps, chicken pox and rubella), the nursing care is probably as important as in that of a case of typhoid fever. In both the hyperpyrexia may demand tepid sponging every half-hour to offset the appearance of convulsions. The bedclothes should be light and the sick-room well ventilated. Personal hygiene also merits attention—frequently in old people the indications for hospitalization are social rather than medical. Bed rest is sometimes difficult to enforce in children or justify to their parents unless the reasons are explained; in post-puberty mumps in boys and in convalescent hepatitis this may be a critical factor in ultimate prognosis. Attention to diet is similarly worth while; glucose for the ketosis of vomiting or high fever is probably more palatable and certainly cheaper when given as a dry powder in a teaspoon than in a proprietary form.

Symptomatic treatment may extend the general practitioner to the limit of his clinical wisdom. Antibiotics have taken the edge off the acumen of many an otherwise astute clinician, for the miraculous properties of these preparations have made life easier for him. Deprived of their therapeutic action, he feels relatively lost and helpless. It is in the virus diseases that nursing and treatment should be combined to the

bad en aspirien of die stoom van pasiënte is standaardvoorskrifte. Vir 'n lastige hoes kan kodeïen gebruik word, maar salisilaat met sy pynstillende en koorswerende eienskappe bly nog die beste geneesmiddel.

Antibiotika en sulfonamides mag nodig wees om die bakteriese komplikasies van masels te genees. In Suid-Afrika is die belangstelling in die profilaktiese gebruik van gamma-globulin slegs akademies maar die gebruik daarvan oorsee dui aan dat dit aan alle kontakte onder die ouderdom van drie jaar gegee moet word as die kind se gesondheid nie goed is nie [2] en seer sekerlik aan alle pasiënte in 'n saal waar 'n geval voorkom. Rooihond is van besonder betekenis omdat dit op 'n vroeë stadium van swangerskap 'n uitwerking op die foetus het, en dit skep derhalwe belangstelling in die moontlikheid van enting teen rooihond soos ook inderdaad teen poliomiëlitis en ander virussiektes, en in „Duitse Masels-teepartytjies" (soos buitelandse skrywers dit bestempel) vir vrouens wat verwag om swanger te word. In kondisies soos herpes zoster en Bornholmsiekte is die verligting van pyn die eerste konsiderasie en hiervoor moet pethidine of selfs morfine gegee word.

Een van die uitstaande kenmerke van virusnavorsing is die gedurige ontdekking van spesifieke etiologiese middels vir die lastige alledaagse kondisies wat in die algemene praktyk aangetref word: die gewone verkoue, appelkoossiekte, epidemiese wintervomering en maaggriep—almal bekende kwale wat nou groot name gekry het. Hierdie ontdekkings dui aan dat virusse altoos aanwesig is en dit is derhalwe dringend noodsaaklik dat mediese navorsing 'n eenvoudige en vinnige metode van diagnose ontdek asook doeltreffende voorbehoedende en terapeutiese middels teen virussiektes.

1. The Practitioner (1954): **173**, 578.
2. McDonald, J. C. and Cockburn, W. C. (1954): Brit. Med. J., **2**, 1076.
3. Annotasie (1954): *Ibid.*, **2**, 1093.

fullest degree. Hot drinks or steam inhalations or a hot bath and aspirin are standard remedies. Codeine may be used in a troublesome cough, but salicylate for its analgesic and antipyretic properties remains the sovereign remedy.

In measles, antibiotics and sulphonamides may be necessary to cure the bacterial complications. The prophylactic use of gamma-globulin remains of academic interest in this country, but its use overseas indicates that it should be given to all contacts under 3 years of age where the health of the child is in any way below normal,[2] and, of course, to all patients in a ward where a case occurs.[3] Rubella has a special importance in view of its action on the foetus in the early months of pregnancy, and interest therefore attaches to the possibility of anti-rubella vaccination (as indeed to vaccination against poliomyelitis and other viral diseases) and to what overseas writers term 'German-measles tea-parties' for women expecting pregnancy. In conditions such as herpes zoster and Bornholm disease, the relief of pain is the paramount consideration and here pethidine or even morphine should be employed.

One of the most striking features of virus research is the constant discovery of specific aetiological agents for troublesome everyday conditions encountered in practice: the common cold, *appelkoossiekte*, epidemic winter vomiting and gastric 'flu—all familiar complaints which are now dignified by longer titles. These discoveries indicate how ever-present the viruses are, and consequently how vitally necessary it is for medical research to perfect simple and rapid means of diagnosis and effective preventive and therapeutic weapons against them.

1. The Practitioner (1954): **173**, 578.
2. McDonald, J. C. and Cockburn, W. C. (1954): Brit. Med. J., **2**, 1076.
3. Annotation (1954): *Ibid.*, **2**, 1093.

THE PROS AND CONS OF TONSILLECTOMY

Operations are notoriously subject to cycles of fashion, and the indications for them equally open to contention. Thus the procedure of tonsillectomy has long been a debatable issue and the decision to operate or not dependent in many cases upon the whims of the individual medical attendant.

There is universal agreement over the specific reasons for the tonsillectomy operation. A genuine case can always be made out for removing chronically infected glandular tissue from any site in the body. It is the general systemic reason which brings the contention. There is the belief on the one hand that the adenotonsillar lymph ring forms a valuable barrier to infection, and on the other hand that the tonsil is a vestigial appendage which, given sufficient time, will always cause trouble. Exponents of the latter view hold that the sooner the glands are removed the better for the individual.

Perhaps because of this reason, tonsillectomy has at one time or another been recommended for almost every disease of childhood, from mental underdevelopment to enuresis. In some parts of the world the operation is carried out on a mass scale—a recent survey in England showed that 6 out of every 10 children entering school were tonsil-less.

Does tonsillectomy do any good? As a prophylactic ritual, probably no; but as a means of relieving blockage of the nose or subjective discomfort not due to some local nasal condition, definitely yes. The size of the tonsils has nothing to do with the question of operation. They may literally dangle down the back of the throat and be quite harmless, provided they are not inflamed or causing mechanical obstruction. Conversely, normal-sized tonsils can be the cause of repeated sore throats, nasal obstruction, earache and other irritating symptoms, which are dramatically relieved by operation.

Three systemic diseases are definitely related to tonsillar infection. In rheumatic fever and acute nephritis the role of tonsillitis in the aetiology has been proved; the question whether the presence of the glands acts

as a physical or natural preventive against anterior poliomyelitis is still unanswered. The accumulated evidence seems to suggest that tonsillectomy does render the person more susceptible to polio, but direct proof of this fact is of course impossible. The most-frequently-quoted example is that of the American family of 6 children, 5 of whom were subjected to tonsillectomy and all contracted polio, 3 fatally; whereas the baby—too young for surgery—actually carried the virus without falling ill of the disease. While these statistics may be misleading, no medical attendant would ever expose a child to the fearful risk of bulbar polio by advising tonsillectomy in the face of an epidemic or an impending one.

It is better to delay operation than to hasten it—something not always easy to justify to an anxious parent. With the use of antibiotics and conservative treatment generally, the need for a potentially dangerous operation for a relatively innocuous condition can often be averted.

Perhaps the most important thing that the doctor can impress upon his patient or the parents is that tonsillectomy is not an inevitable milestone, like the first haircut, but a surgical operation performed only when indicated by definite and specific symptomatology.

In this issue an article by Dr. Wolf Rabkin is published in which he deals exhaustively with this subject from the point of view of the paediatrician.

THE PAEDIATRIC APPROACH TO TONSILLECTOMY

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Complete uniformity about some clear-cut indications for tonsillectomy seems impossible. Finite indications are few. Neither the clinical appearance nor the therapeutic indications can ever be as decisive as, say, in a perforated gastric ulcer or in a coronary thrombosis. It is this lack of definition which led the Editor of the *Year Book of Paediatrics* 1950 to write: 'The common recurrent problem of tonsillectomy and adenoidectomy merits continuing revaluation . . . Harm results both physically and emotionally from indiscriminate removal of lymphoid tissue. The possibility of tonsil removal increases as the number of physicians increases.'

The tonsil is the surgical will-o-the-wisp, although the operation was known as beneficial 2,000 years ago,[1] established successfully a hundred years ago,[2] and so popularized in the present half-century that by now many a practitioner in the Union who lays no claim to surgery firmly asserts, 'I do my own "tonsils" of course,' —which the specialist thoroughly resents.

Tonsillectomy, an apparently simple operation, performed in every nook and corner in the Union or anywhere in the world by any enterprising young practitioner, has led to public debates at the Royal Society of Medicine.[3] The motion and counter-motion read: 'That operations for removal of tonsils are often performed without adequate cause.' 'That this Meeting, whilst deprecating ill-regulated operations, affirms its confidence in the operative removal of tonsils and adenoids as a life-saving and health-giving operation.' This debate did not produce a settled policy about the indications for tonsillectomy.

Operations have their vogue—their advocates and their antagonists—but about tonsils opinions differ very widely indeed. Here is a kaleidoscope of opinions:

Removal of tonsils is always a gamble.[4] In a properly selected case no single operation in Children's Medicine is more successful or shows such dramatic results as that of Tonsillectomy.[5] Another author[6] questions so 'widespread an attack upon a normal structure of the body'. Symptomatic treatment in its most elementary form.[7] In our opinion a large proportion of tonsillectomies now done on children are unnecessary, entail some risk and give no return.[8] From the point of view of prophylaxis of otitis media, cervical adenitis and rheumatic carditis, in any case in which there existed doubt regarding disease of the tonsil, I should recommend that it be removed rather than left.[9] In a report to the Essex Education Committee[10] we read that 'no unsatisfactory result has been seen from conservative treatment in a large number of cases where the tonsils showed definite improvement'. These diverse opinions are reminiscent of the pre-scientific era and serve only to confuse the practitioner. Tonsillectomy removes a diseased organ and eliminates a focus of infection of far-reaching importance, provided always that (*a*) the tonsil and its owner are studied, (*b*) the indications are carefully chosen with patience and without panic, and (*c*) the operation is done efficiently. In spite of the diversity of opinions 200,000 tonsillectomies are performed annually in England and Wales.[11] In the United States tonsillectomy forms one-third of all operations under general anaethesia[11] and it is 3 times as common amongst the well-to-do classes as amongst the poor.

The tonsils are not the only organs in the body that serve as a bait to the surgeon and a haven of refuge to the perplexed practitioner. Other tissues in jeopardy are the 'chronic' appendix and the child's prepuce.

When the appendix is in question, clinical and radiological investigations are carried out; not so with the tonsils; here there is an Alice-in-Wonderland atmosphere. The doctor satisfies himself by saying, 'at any rate, this won't worry us any more', and that in face of the fact that there are 85 deaths a year from tonsillec-

tomy in England and Wales and in the United States 80 deaths a year in children under 15 years of age.[12] There is also a post-operative morbidity which includes lung abscess, pneumonitis, otitis media and mastoiditis. It therefore behoves us to try to clarify the issue.

THE FUNCTIONS OF THE TONSILS AND ADENOIDS

These structures have a function as a unit of the lymphoid tissue of the body and also in relation to their situation in the pathway of deglutition and respiration, but their function is not definitely known, and it is questionable whether they are essential to good health. They are supposed to arrest pathogenic organisms.[13] Clinical assessment is difficult because enlargement of the tonsil may be due to several factors: (*a*) Oral infection e.g., carious teeth; (*b*) a physiological response due to great developmental changes during the period of growth; or (*c*) immunological response to the child's entering an atmosphere of mass infection at school. This occurs between 5 and 7 years and therefore this age-period has the highest incidence of tonsillectomy. Further, in infants and young children lymphoid tissue responds to infection by great swelling and hyperaemia. The general agreement is that the tonsil enlarges between 4 and 6 years. According to Harris [14] the tonsils urgently need attention in the second filling-out period between 7 and 11, when they enlarge appreciably. Schenck's view [15] is that the tonsil is merely a large mass of lymphoid tissue and, because of the vascularity of the contiguous structures, infection spreads to all portions of Waldeyer's ring. The lymph cells are there to remove or to attenuate the organisms and to develop immunity against them. There is evidence that antibodies are formed within the lymph system by phagocytic action and filtration of noxious substances from the circulation by the lymph nodes and the spleen.

Paton's description [6] is worth quoting: 'Solid particles, including bacteria, find their way into the substance of the tonsils from the surface and from the crypts, by what agency is not known. It is believed by some that they are carried in by the lymphocytes. Within the tonsil bacteria may be destroyed by the phagocytic action of polymorphs or endothelial cells, or may persist, when they give rise to foci of disease. When the resisting mechanism of the tonsil breaks down, organisms, or their products, pass by the lymphatics to the tonsillar and lymphatic gland and cause its enlargement. I would therefore call the tonsil a sort of local scavenger. This increases its vulnerability, damages its cellular substance and causes hypertrophy and scar formation. This is aided by the fact that the throat is the site of attack common to most infections. If the tonsil has escaped any infection it will gradually disappear.' Dean [4] similarly postulates that the tonsils are a defence mechanism which swell up in response to an infection and, according to Griffiths,[16] immunity is established by gradual doses of small numbers of organisms. Lederer and Grossman [17] advise waiting until 5 years of age for the operation because the tonsils play a part in auto-vaccination and the defence mechanism. Meyer [18] supports the view that the tonsil is part of the lymphatic defence mechanism against infections and protects the body against the spread of dental, oral and nasal infections. Therefore removal of uninfected tonsils, no matter what size, can be of no benefit and may even be harmful. Wright [19] groups the appendix, Peyer's patches and the constituents of Waldeyer's ring as a part of a generalized sub-epithelial lymphoid tissue which exists in certain places for a purpose, e.g. the adenoid tissue for bacteria in the inspired air, the tonsils for foodstuffs ingested, and the Peyer's patches and the appendix for liquid intestinal contents. According to Wright the tonsils sample the bacterial contents and introduce them into the lymphatic system in the proper amount and at the proper rate for the production of antibodies—an attractive and apparently mechanistic theory. The importance of the tonsils is emphasized by Paton because of the following: (1) They have a free blood supply from 3 sources—dorsalis linguae (lingual), ascending palatine (facial), and descending palatine (maxillary) and (2) a double nerve supply, (3) they occur only in mammals, (4) they are especially active during childhood, (5) they contain many germinal centres for the production of lymphocytes, and (6) their removal is followed by hypertrophy of the neighbouring lymphoid tissue.

THE PATHOLOGY OF THE TONSILS AND ADENOIDS

Because there is a considerable variation in the appearance of the inflamed tonsil, it is impossible to give a finite description, as say of the drum or of the nasal passages. Further, the tonsil is, by its very situation, often studied only perfunctorily. One listens to the heart and lungs for some considerable time, palpates an abdomen most carefully, and tries to elicit a reflex several times, but looking at a tonsil is a matter of moments. There is no time in a small child to study it with ease. The tonsil is soon covered up with post-nasal discharge, mucus, saliva and often vomitus. It is therefore incumbent upon us to choose our criteria carefully. We put forward certain assumptions and we ask specific questions:

(*a*) Those living under good conditions are usually subject to less infection than the poor.

(*b*) The tonsil is more commonly attacked in many diseases in infancy and childhood than any other organ in the body. It is recognized as the most frequent portal of entry for pathogenic agents and it must therefore be damaged in the process.

(*c*) The size and colour of the tonsils are often the hurried criterion used for deciding for or against tonsillectomy. Paton says enlargement alone is no proof of disease but is due to the activity of the germinal follicles. He estimates the size in 6 degrees of visibility in relation to the anterior pillar.

(*d*) The tonsil, normally the policeman of the throat, is dangerous when it succumbs to infection.

To study the pathology of the tonsil itself needs a most careful assessment of its naked eye appearance, its relationship to the pillars, and the presence of small rounded puckered areas denoting the ravages of previous

inflammation. Smegma-like substance expressed from the crypts should be examined microscopically or it will be misinterpreted as pus. Redness of the pillars is a common finding but should not be considered pathological unless the redness and congestion are noted on several occasions. This is important, because inflammation may be present without pain or discomfort and *vice versa*. Sometimes there is a history of sore throat or tonsillitis and we may have to judge either by the appearance of the pharynx or by the sequelae—nephritis or acute rheumatism.

Layton [21] defines septic tonsils as those in which 'there are living in the crypts organisms which are doing harm locally or to the body generally by absorption into the blood stream'. A more recent reference [21] quotes Rummelkamp's description—pictorial and factual—of a typical group-A-B haemolytic streptococcal infection: 'There is a relatively rapid onset with soreness on swallowing . . . general swelling of the lymphoid tissue of the pharynx and bright red inflammation of the faucial mucosa. These discrete or confluent patches of yellow exudate stand out against the red and congested background. There may be considerable variations but the more severe throats are those caused by group-A-B haemolytic streptococcus.' It is interesting to recall some earlier views. Epstein [22] has declared that the dictum that the tonsil must come out when infected, is open to 2 objections: (*a*) It has no relationship to children because in them the tonsils are usually removed because of the infection in the upper respiratory tract. (*b*) We do not know when a tonsil is infected—it is not even certain from microscopic examination 'let alone clinical examination'. In our opinion, the history of acute tonsillitis is a more reliable indication of chronic infection than the appearance of the tonsils.

Breuning [23] warns against the small, hard, pale gland which may appear normal but which when removed contains pockets of pus. He also incriminates the dusky red tonsil, with crypts in the upper third from which can be squeezed a milky secretion. The subacute tonsil is 'very large, dull-red and spongy, the anterior pillars are congested and the palate assumes a deep red colour'. Cheeseman [24] classifies tonsillitis as (1) primary from direct contact—infected milk, water or food, and also part of a general infection, e.g., scarlet fever or measles; and (2) secondary from septic teeth or nasal suppuration. He considers the tonsil as a lymphoid gland; it has a structure like a lymphoid gland and behaves like one. Because of its exposed position it is susceptible to attacks of inflammation.

Lederer [17] says, 'The theory that the tonsils are a common site for focal infection is not "completely accepted" '. He states (rather stoically) that there is nothing to show with certainty that tonsillectomy causes or prevents disease. De Veer, on the other hand, says that abscessed crypts are practically universal beyond infancy. Dean [4] maintains that he could not identify a chronically infected tonsil except after removal and microscopic study—a parodoxical attitude reminiscent of Glover's reference to a saying at Guy's, 'Call no man healthy until he is dead and Dr. Moxon has performed a *post mortem*'.

Criteria in Examination

Look at the throat and note:

1. *Size of tonsil.* Appearance and state of the pillars and their relationship to the tonsils. Any adhesions?
2. *Colour.* Pale, red or purple? Bright daylight, in our opinion, is better than artificial light.
3. *Consistency.* Sclerotic and hard, or soft and spongy? This is especially important after measles or scarlet fever, when they may remain large and unhealthy for a considerable time and then may shrivel into the hard fibrotic type.
4. Squeezing the tonsils with the spatula. This may reveal milky or creamy odoriferous substance consisting of debris or pus.
5. See if the post-pharyngeal wall is granular or not.
6. Enlarged glands. Lymphatic glands must be looked for, especially in fat children. Enlarged upper cervical glands are common in post-nasal and scalp infection, but the extra-tonsillar gland is an indication of the state of the tonsils. Note the site of the particular gland, its consistency, its mobility and the presence of periglandular enlargement. Look carefully for dental caries.[25]
7. Examine the sinuses. Griffiths [25] showed that dyes injected into the sinuses are recovered in the tonsil, i.e. the tonsils act as a filter for toxins produced in the sinuses.
8. The examination of the mouth and pharynx, nose and ears can be done by any general practitioner. Always inquire into any signs of Eustachian blockage.

THE TONSIL IN RELATION TO CERTAIN DISEASES

1. *Rheumatic Fever.* It is commonly held that rheumatic fever is a complication of an infection of the throat of a susceptible individual by group-A-B haemolytic streptococcus and that 'no other organism gives rise to acute rheumatic fever and that all patients with acute rheumatic fever have had antecedent streptococcal throat infections'.[26] Fifty per cent of schoolboys with subacute rheumatism had a history of an antecedent sore throat and 17·5% of these had also muscular rheumatism.[24] Green [27] confirms this finding in 78% of rheumatic cases: there was a history of a throat infection 3 weeks before; 60% were positive for a haemolytic streptococcus, which in the majority of cases proved on culture to be A-B strains. In 200 rheumatic and 200 non-rheumatic cases he recovered a haemolytic streptococcus from 58% of the former and 30% of the latter.

There seems to be an inverse relationship between the prophylactic effect of sulphonamide, at blood level of 40 micrograms, and the incidence of rheumatic recrudescence.

Coburn [28] explains the pathogenesis of rheumatic fever along these lines: (1) Products of haemolytic streptococci are distributed widely throughout the body at the time of acute infection. (2) Body tissues combine with these toxins to form antigens. (3) There follows a period of formation of antibody against the tissue-toxin complex. (4) When the titre is sufficiently high an antigen-antibody reaction occurs.

Bradley [29] suggests that a considerable proportion of people are predisposed to rheumatism, and that it is not impracticable to protect genetically predisposed individuals in contact with *Streptococcus pyogenes* by means of chemoprophylaxis. In this he is in the minority; he also suggests restricting the size of the family in these cases. Rantz *et al.*[30] tried to show that the latent interval between sore throat and rheumatic fever is not really latent as shown by the sedimentation rate and electrocardiogram. They suggest that rheumatic fever is really part of a post-streptococcic state, and they go further and postulate that rheumatic fever, post-scarlatinal arthritis and atypical rheumatic fever could be regarded as a single entity. Schlesinger [31] suggested many years ago an antigen-antibody reaction and that a second infection is the exciting cause of the rheumatic state. Illingworth [32] thinks there is no evidence to show that tonsillectomy has proved of any value in rheumatism, though rheumatic relapses are largely brought about by this sort of infection. Rolleston [33] in an analysis of 1,136 cases found that 9·8% had a history that suggested rheumatism but, all in all, they were not more prone to rheumatism when they had enlarged tonsils and adenoids. On the other hand Silkowsky *et al.*[34], in an epidemic of rheumatic fever in a children's institution following an outbreak of tonsillitis with a haemolytic streptococcus in the throat, found that in 23 of the 88 cases there was a latent period of 10-23 days between the tonsillitis and rheumatic fever. Poynton and Schlesinger [35] say: 'Eneucleation of the tonsils certainly tends to remove throat infections which on each occasion lay each rheumatic patient open to a recrudescence of the disease.'

2. *Nephritis.* We know that during an attack of tonsillitis the urine may contain blood, albumin and casts. It is important to look for a focus of infection, especially in the tonsil. Willcox [36] mentions that rheumatism or nephritis are often ushered in or accompanied by tonsillitis. In scarlet fever streptococci are found in the urine and in furunculosis staphylococci. Illingworth [32] lays it down as a rule that if tonsils are removed because of acute nephritis or rheumatic fever or any other condition related to a haemolytic process, only tonsils showing evidence of infection should be removed. The mere presence of a haemolytic streptococcus in the throat may mean only a carrier state as in diphtheria carriers. He questions the value of tonsillectomy and concludes from a study of 301 nephritic patients of whom 20·2% had tonsillectomy done that tonsillectomy (1) may cause nephritis, (2) does not cure nephritis, (3) does not prevent nephritis, and (4) does not prevent it from progressing to a chronic stage. Collier [37] says, 'Even when the history indicates a relation between tonsillar function and onset of nephritis, removal of the tonsils does not appear to influence the course of the disease'.

Lyttle [38] suggests that it would be reasonable to remove tonsils as a precautionary measure against nephritis, but nephritis itself should not be considered as an indication for tonsillectomy. He suggests that it is an elective operation, i.e. each case should be judged on its own merits, and that the operation should not be done until all signs of acute nephritis have subsided. Kaiser [39] on the other hand maintains that tonsils do not play a big role in the causation and development of nephritis.

3. *Upper Respiratory Tract Infection.* There is general agreement that early removal of the tonsil and adenoids increases the susceptibility to infection of the respiratory tract. Certainly bronchitis and pneumonia are not prevented in tonsillectomized children. Tonsillectomy offers no radical solution to the common cold, otitis media, sinusitis, or laryngitis, which have a devitalizing influence upon the growing child. Kaiser [39] is of opinion that tonsillectomy prevents upper respiratory infection but the 'presence of tonsils may inhibit a lower-pulmonary-tract infection'. He is convinced that tonsillectomy cures recurrent tonsillitis, cervical adenitis, otitis media and colds. According to Willcox [36] the chronically-infected tonsil serves as a focus of infection causing rhinitis, sinusitis and pharyngitis. This is contrary to Illingworth, who believes that tonsils should not be removed for frequent colds, upper respiratory infections or allergic conditions.

4. *Poliomyelitis.* Many years ago Fillerman and Fischer [40] suggested that traumatized adenoid and tonsillar tissue might afford a portal of entry for the virus of poliomyelitis in some cases. If it does occur, it is the bulbar form. Moller [41] suggested postponement of all tonsillectomies in the presence of an epidemic because of a report [42] of the occurrence of poliomyelitis in 5 members of a family following tonsillectomy. This has shaken many an authority, and is very difficult to oppose. Anderson [43] records that during an epidemic of poliomyelitis in the State of Utah a questionnaire was sent to 334 doctors in regard to tonsillectomies and poliomyelitis. The answers showed (amongst other facts) that 43% of the bulbar and bulbospinal cases were preceded by a tonsillectomy within 30 days of the onset. The incidence of this form of poliomyelitis was 16 times greater in the tonsillectomized children than in the general population. After considering the divergent opinions it seems that a causal relationship has been established between adeno-tonsillectomy and poliomyelitis. We should act on that until it is disproved.

5. *Diphtheria.* Most people are of the opinion that diphtheria and scarlet fever are more severe in children with enlarged tonsils and adenoids. Feingold [44] agrees with that view. It has certainly been our personal experience. *C. diphtheriae* has been found in the throat secretions of children who had tonsillectomy done 8 weeks after having had diphtheria.

6. *Allergy and Asthma.* The reaction of the allergic child is governed by the type of infection:

(*a*) In an upper-respiratory-tract infection, e.g. tonsillitis or sinusitis, the allergy will be quiescent in the invasion period and aggravated at the height of infection.

(*b*) In the infectious diseases, e.g. measles and scarlet fever, the allergic state improves at the height of infection.

Repeated or chronic infections worsen the allergy and lower the effect of the therapy both upon the allergy and

the infection. Each re-infection influences the allergy and raises its base line. Because the tonsil is a focus of infection, it is best to eradicate it.

However, in asthma—which must be precisely differentiated from an upper-respiratory-tract infection—it is best to delay the removal of the tonsils, which gives us time for reflection to decide whether or no there is a respiratory allergy. This is clearly explained diagramatically by Feingold.[45]

7. *Abdominal Pain.* It is impossible now to go into the question of abdominal pain in relation to tonsillitis. There is no doubt, however, that ileo-caecal adenitis produces abdominal pain. We are all familiar with abdominal pain in scarlet fever and particularly in rheumatic fever, where, after 2-3 days in hospital, a mitral diastolic murmur often appears. Careful clinical assessment is invaluable in these cases.

THE INDICATIONS FOR TONSILLECTOMY AND ADENOIDECTOMY

The tonsil has been looked upon as a pathological vehicle for inflammation in the respiratory mucous membrane—of the mouth, nose and bronchi, including the sinuses and the ears—causing nasal obstruction, sinusitis, pharyngitis, coughs and colds, bronchitis, otitis media and mastoiditis. Children with dysfunctions such as night terrors, nocturnal enuresis, allergy and asthma, and habit spasm, have been associated in the physician's mind with enlarged tonsils and adenoids. The tonsil is being blamed for tuberculous adenitis and, if it becomes infected with specific organisms, it may cause such crippling diseases as rheumatic fever or nephritis. Quinsy is an obvious local infection. King [21] mentions that over half of 150 cases of coronary occlusion had repeated attacks of tonsillitis or quinsy. Hypertension has been given as a reason for tonsillectomy.[47] Is it possible that this simple physiological structure can become so Machiavellian? Are we right in removing an organ at the slightest sign of abnormality—as is being done so commonly with the young child's prepuce? What criteria must we use to sift the grain from the chaff? Should the criteria be clinical or bacteriological? Can we ever expect our criteria to be as definite as those used for doing a lobectomy or a gastrectomy? Tonsillectomy is a major operation and has a certain mortality and post-operative sequelae. The criteria must be properly assessed. We can classify them as (A) positive indications for tonsillectomy, (B) probable indications and (C) presumptive indications.

A. Positive Indications

(*a*) *Repeated attacks* of tonsillitis *per se*—with or without cervical adenitis. This is subject to one proviso—that the tonsils are examined when they are inflamed and again in the interval. This has been our routine for many years.

(*b*) After the occurrence of *quinsy*. Nobody will gainsay these two indications.

(*c*) *Hypertrophy* with *obstruction* which produces *symptoms*. This is rare. One has often seen the tonsils almost touching each other, with no ill effect. Collier [37] says: 'Occasionally in young children prominent tonsils projecting into the oro-pharynx, or in contact with the posterior pharyngeal wall, interfere sufficiently to give the child a disinclination for eating that hinders his normal nutrition. As soon as the congestion subsides, the tonsils shrink in size.'

(*d*) *Evidence of persistent infection of the peritonsillar tissues* when there is glandular enlargement and no obvious signs of disease. But an acute infection is no indication for operation.

(*e*) *Chronic diphtheria carriers*, where all other medication has failed.

(*f*) *Tuberculous adenitis.* Removal of the glands should be performed first. (The tonsils in these cases are shown to have tuberculous giant-cell systems.) If tonsillectomy is done first and there is a mild post-operative infection of the tonsillar bed, it may lead to a breaking down of the glands and abscess formation.[48]

B. Probable Indications

(*a*) Recurrent attacks of otitis media
(*b*) Repeated attacks of pharyngitis
(*c*) Rheumatic Fever
(*d*) Nephritis

C. Presumptive Indications

(*a*) *Repeated coughs and colds.* This is a matter for personal judgment. The tonsils should be examined at the height of the attack and again in the interval.

(*b*) *Chronic nasal catarrh.* The sinuses should be examined as a possible cause. Sinusitis in children is no easy problem. Brown [49] lists 50 symptoms of chronic sinusitis in children with a differential diagnosis of each symptom! He says tonsils and adenoids have no relation to frequent or prolonged colds. Tonsillectomy, in our opinion, neither prevents colds nor lowers their incidence.

(*c*) *Bronchitis.* This seems to be no justification for tonsillectomy.

(*d*) *As a prophylactic.* Tonsillectomy is of no value as a prophylactic except possibly in diphtheria. It is suggested that diphtheria (and scarlet fever) is a much milder disease in tonsillectomized children. In diphtheria there is less chance of a carrier state. Cameron[50] thinks tonsillectomy may have to be done for the sake of the mother if there are repeated infections. It is pertinent to recall several statements made by Layton.[20] He suggests that if there are 2 attacks with joint involvement or 3 without, due to micro-organisms within the tonsils, the tonsils and adenoids should be removed. He is against tonsillectomy if there is a risk of measles because if the child develops measles it may lead to otitis media and mastoiditis. Layton is a humanist. He is not concerned with the wealthy whose life is blessed with good hygiene. He is anxious about the poor and is averse to removing a barrier against infection. He adds: 'No child living under conditions of overcrowding should ever have the tonsils removed if it can possibly be avoided.' Layton is against removing tonsils in children under 5 years of age. Removal of lymphoid tissue from the naso-pharynx diminishes the resistance to general infections—colds, sore throats, etc. In our opinion the tonsils should not be removed below the age of 6 or 7 except in special circumstances;

this has been our practice for many years, subject, of course, to assessment of the individual case. Gale [46] advises removal after 5 years of age but he removes adenoids at any age if there is nasal or Eustachian-tube obstruction. Adenoidectomy has been done in the very young because of recent or chronic infection of the sinuses.

CONCLUSION

In the light of what has been said and in view of the long catalogue of sins committed by, or through, the tonsil, what should be the criteria for its removal? The criteria should be clear and finite, as are the indications on which an abdominal case, for example, must be judged. With a proper assessment of each case as delineated, the practitioner should be on fairly sound ground.

Permit me for a moment to make a plea for the unfortunate child. It is a *sine qua non* that each patient must get the benefit of modern scientific investigation and treatment, including psychological care and follow-up. The very rich receive that benefit and some say the very poor, though this does not hold in tonsillectomy. The method adopted on a tonsil morning is reminiscent of the barber's chair—mysterious and exciting to a small boy who for the first time is having a hair-cut.

The children are lined up waiting, patiently waiting. The child finds some comfort—possibly a little fun—at seeing many others in the same position as himself, but to the sympathetic observer he is obviously in fear and dread of what is about to be done to him. To be taken away by a strange nurse and porter, to be received by another strange nurse in the awesome atmosphere of the operating theatre or anteroom, arouses fear of the unknown. The little victim's feelings are best described by the title Churchill chose for one of the volumes of his *Second World War*—'Alone'. He is whisked away to take his turn upon a table in a darkened room and confronted by a spectacled man in a white gown and large mirror on his forehead. He is barely taking all this in when the mask is clamped on his face.

On regaining consciousness he has a deadened sensation in his throat, a rawness in the posterior nasopharynx and a blocked and bleeding nose. Do we ever stop to think of the psychological trauma we have inflicted? No other operation in a hospital, except a circumcision, is done in such a hasty and unseemly manner. The child has much to remember and he remembers for a long time.

The obvious remedy is to treat the child as an in-patient, tell him something about the sensation he will experience, and give him full premedication. One hopes that chemotherapy and the antibiotics may reduce the necessity for this ritual.

It is easy to be overawed by a Report of the Medical Research Council which says: 'It is a little difficult to believe that of the mass of tonsillectomy performed today, all subjects for operation are selected with true discrimination, and one cannot avoid the conclusion that there is a tendency for the operation to be performed as a routine prophylactic ritual for no particular reason and with no particular result.' But this is not a condemnation of tonsillectomy. It is rather an exhortation to do what is essential in medicine—to judge each case on its own merits and, possibly with some help, to establish the diagnosis. This applies to all of us—the general practitioner, the paediatrician and the ear, nose and throat surgeon. The operation is being performed on about a third of the European child population, and the time is opportune to provoke thoughtful consideration of the subject. In this way we shall 'add life to years and not merely years to life'.

POSTSCRIPT

Since this paper was written (1952), Eckenhoff [a]) has investigated the post-operative mental state of young children after otolaryngologic operations under anaesthesia and found 17% showed personality alterations in varying degrees—night terrors, temper tantrums, fear phenomena and bed-wetting. An editorial in the *British Medical Journal* [b]) advises the anaesthetist to mingle kindness with pharmacology. MacKeith, [c]) whose article abounds in kindness and common sense, says aptly and correctly that being trundled on a trolley lying flat so that one cannot see where one is going, is alarming. It is encouraging to read in a contribution [d]) from the Harvard Medical School to a symposium on the tonsil-adenoid question that the emotional trauma of years ago has been removed as physicians have improved their knowledge of the psychologic effect of hospitalization on the child.

In a recent article on psychological care Oldsaker [e]) supplements his sound advice on the approach to the child by a story—pictorial and poetic—with which to condition the child for the operation. To quote two stanzas:

> Although his throat was feeling sore,
> Tom was no different from before,
> That night he had a happy treat,
> All the ice cream he could eat.
>
> Soon Tom was back in his own bed,
> He played with brother and he said,
> Freddie, you can look and see,
> I had my Tonsillectomy.

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PRIMÊRE KARSINOOM VAN DIE TUBA UTERINA MET 'N VERSLAG VAN 'N GEVAL PRE-OPERATIEF GEDIAGNOSEER

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Primêre karsinoom van die tuba uterina is die mees seldsame vorm van ginekologiese karsinoom, en 'n ginekoloog mag gedurende sy hele professionele loopbaan nooit 'n enkele geval teëkom nie.[1] Vanaf 1888 toe Orthmann en Doran elk 'n onbetwisbare geval beskryf het, tot aan die begin van 1954, is ongeveer 490 gevalle in die literatuur beskryf;[2] sommige van hierdie gevalle was twyfelagtig, en menige ander gevalle het seker nooit hul pad na die literatuur gevind nie. Dat die toestand seldsaam is, is nie te betwyfel nie. In 45 jaar (1903 tot 1948) het by die ‚Free Hospital for Women', Brookline, Mass., onder 90,611 ginekologiese opnames en 3,878 primêre maligniteite van die genitale weë net 12 gevalle van primêre karsinoom van die buis voorgekom.[3]

Hierdie verslag word gedoen vir twee redes: eerstens omdat slegs enkele gevalle van die toestand nog ooit pre-operatief gediagnoseer is, en tweedens omdat Way (1951)[4] die belangrikheid beklemtoon van alle gevalle wat teëgekom word te beskryf: ‚This, the rarest of gynaecological cancer, will on account of its rarity remain for long years to come an enigma in many respects. It does however show the importance of documenting well and reporting every case that one encounters.'

Van die gevalle wat in die literatuur aangehaal is, kan wel sommige gevolgtrekkings gemaak word. Besonder treffend is die bevinding dat die diagnose feitlik nooit pre-operatief gemaak word nie, selfs by operasie soms gemis word, en dat die gevalle uiters laat geopereer word met gevolglik 'n slegte prognose. Indien die toestand in gedagte gehou word, mag gevalle meer dikwels gevind word.

Voorkoms-syfer. By die Mayo-Kliniek[5] was daar, oor 'n tydperk van 40 jaar, 16 gevalle onder 10,000 gevalle van ginekologiese maligniteit, d.w.s. 0·16% van ginekologiese primêre maligne toestande. Ander skrywers stel die voorkoms op 0·3% tot 0·6%.

Ouderdoms-syfer. Die toestand kom op enige ouderdom voor tussen 18 en 80 jaar, maar meeste van die pasiënte is tussen 40 en 65 jaar oud. Ongeveer eenderde van die pasiënte het nooit kinders gehad nie.

Simptome en tekens. Klagtes is dikwels gering sodat die toestand gevorderd is by die tyd dat die pasiënt gesien word. Nie enige van die simptome en tekens is konstant nie en kan ook nie as kenmerkend beskou word nie. 'n Bloederige of serosanguineuse ongereelde vaginale afskeiding is soms (ongeveer 10% van gevalle) teenwoordig;[4, 6, 7] abdominale koliek gevolg deur 'n waterige afskeiding per vaginam word aangehaal asook hydrops tubae profluens. Laasgenoemde egter het slegs in een van die 12 gevalle van Hu, Taymor en Hertig[3] voorgekom. Ander klagtes is gewigsverlies en vergroting van die buik. Pyn is veronderstel om vroeër voor te kom as in gevalle van karsinoom van die corpus uteri en is die gevolg van spanning van die tuba-wand. By ondersoek is die mees konstante bevinding 'n tumor in die onderbuik en word gevind in 85% van die gevalle. Ascitis is seldsaam en kom alleenlik in die laat stadia voor. Die rol van histerosalpingografie en vaginale smere in die vroeë diagnose word later gemeld.

Diagnose. Onder foutiewe pre-operatiewe diagnoses wat mees dikwels gemaak word, kan genoem word miomata,[6, 8] ovariale kiestes en neoplasmata (vernaamlik ovariale karsinoom in die laat gevalle), uterine karsinoom, endometriose, hidro-, pio-, en hematosalpinx, tubo-ovariale absesse, en tuberkulose van die buise en peritoneum. Selfs by operasie gebeur dit soms

dat net 'n buis alleen verwyder word deurdat die letsel aangesien word vir 'n inflammatoriese toestand.[9] Waar by operasie verspreide abdominale karsinomatose gevind word, mag dit onmoontlik wees om 'n primêre karsinoom van die tuba met verspreiding te onderskei van 'n primêre karsinoom van die ovarium met verspreiding, en aangesien laasgenoemde meer dikwels voorkom, word hierdie gevalle dan gewoonlik as sulks beskou. Op hierdie manier word sommige van die gevalle nooit herken nie.

Way (1951)[4] beweer dat 'n akkurate diagnose nog nooit pre-operatief gemaak is nie. Stern en Hanley[7] het egter gevind dat in 2 uit die laaste 61 gevalle wat in Noord-Amerika gerapporteer is, die toestand korrek pre-operatief gediagnoseer is. McGoldrick (1943)[10] het onder 376 gevalle in die literatuur net een gevind wat gediagnoseer was en dit was die geval van Marzloff[11] in 1940. 'n Geval is wel deur Falk[12] in 1898 beskryf waar hy die diagnose gemaak het van materiaal wat hy deur posterior kolpotomie-punksie afgetrek het.

Salpingografie word deur sommige skrywers sterk aanbeveel en 2 gevalle is op dié manier pre-operatief gediagnoseer (Antonowitsch[13]). Marzloff is 'n groot voorstander vir dié metode en beweer dat dit onskadelik is aangesien die fimbriale einde van die buis gewoonlik vroeg reeds afgesluit het sodat die metode nie peritoneale disseminasie kan teweegbring nie. Meeste skrywers is egter daarteen en beskou dit as definitief gevaarlik daar die fimbriale einde nie *altyd* toe is nie, en verder beskou hulle dit van min waarde te wees.

Vaginale smere word erken van hulp te wees in die vroeëre diagnose van die toestand (Cereser,[6] Emge,[14] Hu,[3] Ayre[9]). Positiewe vaginale smere wat adenokarsinoom aandui tesame met 'n negatiewe curettage vir endometriële karsinoom is suggestief van karsinoom van die tuba uterina.[15] McClean[16] het die gebruik van 'n plastiese doppie aanbeveel met die doel om selle van die boonste genitale dele op te vang en te konsentreer vir ondersoek; hy rapporteer 'n geval waar 'n vroeë tubale maligniteit op dié manier korrek pre-operatief gediagnoseer is. Maligne selle kan in die vaginale smere voorkom as gevolg van 'n karsinoom van die ovarium maar die kanse hiervoor is veel geringer en dit vereis dat die tumor oppervlakkig moet wees, of dat dit die kapsel geperforeer het met loslating van maligne selle in die buikholte, of dat die ovariale gewas reeds na die buis of uterusholte uitgesaai het. Ayre meen dat die enigste rede waarom karsinoom van die tuba nie meer dikwels al deur vaginale smere ontdek is nie, is dat die toestand so uiters seldsaam is.

Die kriteria vir die erkenning van 'n geval as 'n primêre karsinoom van die buis ná operasie kan as volg opgesom word (Weekes en Whiting):[17] (*a*) die hoof tumor moet in die buis wees; (*b*) geen primêre letsel moet in die ovarium, uterus of ander organe gevind word nie; en (*c*) mikroskopies is die tubale slymvlies hoofsaaklik aangetas en 'n verandering van die tubale slymvlies van die normaal na maligne epiteel moet demonstreer word.

Patologie. Hierdie tumore is almal adenokarsinoom alhoewel plaveisel metaplasie gerapporteer is. Makroskopies is die gewas gewoonlik papillêr maar areas van soliede aard kan gesien word. Die tuba is uitgesit en kan 'n besondere groot tumor vorm. Die karsinoom is in feitlik alle gevalle beperk tot die buitenste tweederdes van die buis en neig na die fimbriale end. Deurbreking van die buis-oppervlakte vind byna altyd uiteindelik plaas. Die tumor is bilateraal in ongeveer een-derde van gevalle. Wanneer die tumor oopgesny word soos altyd gedoen behoort te word waar enige abnormale buis by operasie verwyder word, dan het die snyoppervlakte 'n grys papillêre voorkoms. Dikwels is daar meegaande adneksale siektetoestande teenwoordig, bv. chroniese salpingitis of ovariale kiestes.

Mikroskopies word twee vorms beskryf, nl. papillêr an alveolêr. Hu en medewerkers beskou die alveolêre vorm as 'n laat stadium van die fundamentele papillêre patroon. Die toestand is soms geassosieerd met tuberkulose van die buis,[8, 17, 18] en aangesien die twee toestande ook hier histologies mekaar kan simuleer mag dit tot verdere verwarring lei.

Verspreiding van tubale karsinoom is hoofsaaklik na die peritoneum deur die fimbriale ostium of buiswand; verdere verspreiding is soos dié van ovariale karsinoom; limfklier-metastases ontstaan langs die arteria iliaca communis en strek hoër op langs die groot bloedvate; bloedverspreiding na die lewer en longe is beskryf.

Behandeling. Die erkende vorm van behandeling is 'n radikale panhisterektomie wat verwydering van beide adnexae insluit, gevolg deur diep X-straal-behandeling. Om die uterus agter te laat om te dien as 'n houer vir radiumaanwending soos by die Radiumhemmet gedoen is,[4] dra nie algemene goedkeuring weg nie. Die Wertheim-operasie word ook genoem as behandeling.[1]

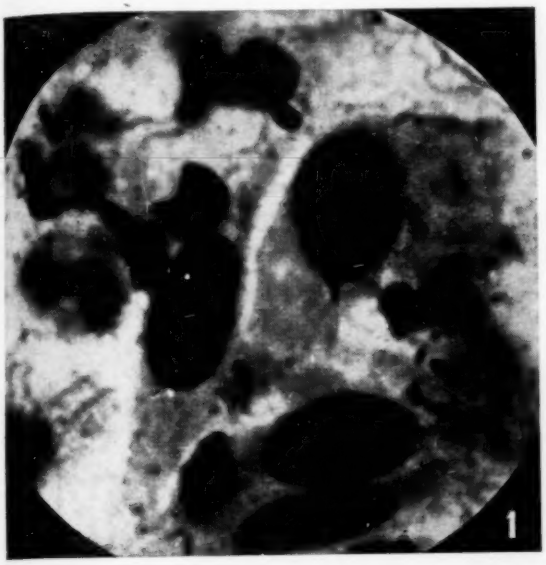
Die prognose is oor die algemeen swak omdat die toestand gewoonlik eers laat ontdek word. Die toestand bly egter relatief lank gelokaliseerd mits die fimbriale end van die buis toegaan voordat maligne selle afbreek; die wand van die buis laat ook gewoonlik 'n groot mate van uitrekking toe voordat die spierlae deurdring word. Die beste resultate wat beskryf is, is die van Hu en medewerkers (1950) wat 12 gevalle beskryf het met 'n 5-jaar herstel van 40%, insluitend een pasiënt wat lewe na 28 jaar. Die beter gedifferensieerde papillêre vorms hou 'n beter prognose in.[3]

VERSLAG VAN GEVAL

H.v.S., 'n blanke vrou, 74 jaar oud, para II, is opgeneem op 24 Mei 1949 met die klagtes van bloederige afskeiding *per vaginam* vir 6 maande voor opname en vae pyne in die onderbuik; sy het ook gekla van pyne in die ledemate. Die vaginale bloeding was afwisselend in die begin maar het later aanhoudend geword. Drie maande voor opname is 'n curettage gedoen vir die bloeding en 'n endometriële poliep is gevind maar geen teken van maligniteit nie. Daarna was die bloeding tydelik beter. Sy het geen ander operasies of ernstige siektes gehad nie.

Algemene ondersoek het niks van belang opgelewer nie, behalwe 'n mate van teerheid oor die onderbuik. By vaginale ondersoek is 'n geringe hoeveelheid rooi bloederige afskeiding vanuit die servikale os opgemerk. Die uterus was normaal maar 'n geswel kon regs van die uterus gevoel word. Vaginale smere is geneem en was positief vir karsinoom (Fig. 1 en 2). Die curettage is herhaal en geen weefsel is verkry vir histologiese ondersoek nie. Onder narkose kon 'n geswel die grootte van 'n hoendereier in die gebied van die regter adnexa gevoel word.

Operasie. Op 27 Mei 1949 is laparotomie uitgevoer en 'n klein normale uterus gevind terwyl daar 'n groot worsvormige uitsetting van die regter tuba uterina teenwoordig was. Beide ovaria was atrofies. 'n Totale histerektomie met bilaterale verwydering van die adnexae is uitgevoer. Geen vergrote kliere is in die buikholte gevind nie. Post-operatiewe herstel was sonder teenspoed en die pasiënt is uit die hospitaal ontslaan op 19 Junie 1949.



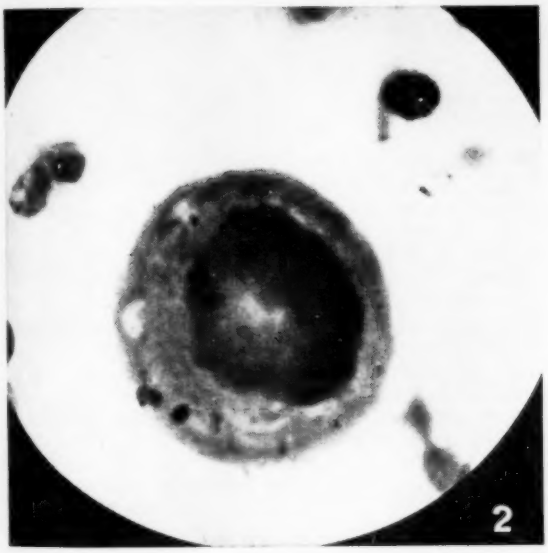


Fig. 1 *en* 2. Maligne-selle gevind by vaginale smeer-ondersoek in die geval van primêre karsinoom van die tuba uterina hier beskryf (Papanicolaou-kleurmetode hoë vergroting).

Weens haar ouderdom en omrede van die beperktheid van die letsel is besluit om nie post-operatiewe bestraling toe te dien nie.

Die operasiemonster. Die uterus, ovaria en linker buis was normaal. Die regter buis was uitgeset in die laterale twee-derde deel, worsvormig, en die fimbriale end was afgesluit (Fig. 3). Die buis is oopgesny en gevind gevul te wees met 'n papillêre gewas. Histologiese snitte het 'n papillêre adenokarsinoom van die regter tuba uterina getoon: daar was makroskopies en mikroskopies geen belangrike veranderinge in die ovaria of uterus nie.

Opvolg. Van 'n kollega is verneem dat die pasiënt wel was 18 maande na die operasie, maar verdere opvolg was nie moontlik nie.

BESPREKING

Hierdie geval beklemtoon weereens die belang en die waarde van die vaginale smeertegniek in die vroeë diagnose van ginekologiese karsinoom, daar dit lank voor die ander metodes van ondersoek 'n positiewe indikasie van maligniteit mag gee.

Alhoewel dit nie moontlik was om ander siektetoestande pre-operatief met sekerheid uit te skakel nie, was die diagnose van tubale karsinoom hier gemaak op grond van die teenwoordigheid van 'n adneksale tumor, 'n positiewe Papanicolaou-smeer vir karsinoom, en negatiewe curettages van die uterusholte. Ander toestande wat dieselfde bevindings mag gee is: (*a*) 'n

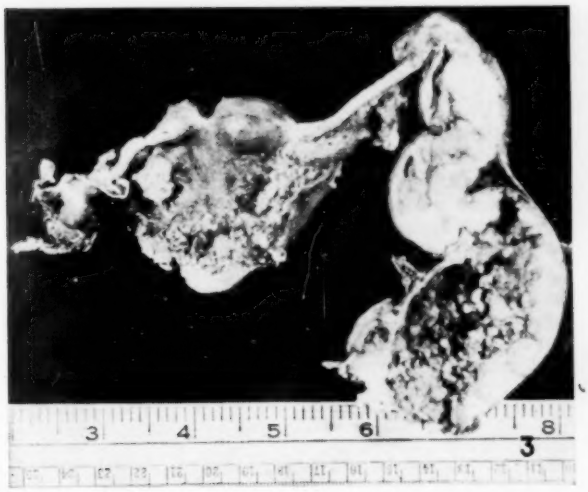


Fig. 3. Foto van die uterus en adnexa wat by operasie verwyder is. Posterior aspek van die regter buis is oopgesny en wys die papillêre voorkoms van die karsinoom.

klein karsinoom in 'n cornua van die uterusholte wat gemis word met curettage, plus 'n adneksale geswel, of (*b*) 'n karsinoom van die ovarium met verspreiding na die buis of uterus.

Hierdie geval dui ook aan hoe belangrik dit is om alle pasiënte wat post-menopousaal weer begin bloei na 'n negatiewe curettage, waar moontlik liewer te opereer. Ook behoort enige onverklaarbare adneksale massa chirurgies ondersoek te word indien die pasiënt se algemene toestand dit toelaat.

OPSOMMING

'n Oorsig word gegee van primêre karsinoom van die tuba uterina, en 'n geval word beskryf waar die diagnose pre-operatief gemaak is met behulp van die vaginale smeermetode.

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ANAESTHESIA FOR BRONCHOSCOPY: USE OF RESPIRATOR

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Before the introduction of the muscle-relaxants the methods available for bronchoscopy were (*a*) local analgesia and (*b*) deep general anaesthesia.

(*a*) *Local analgesia* is very unpleasant and trying for the patient. It carries the danger of death from sensitivity or overdosage of the analgesic used.

(*b*) *The general anaesthesia* required is full third-plane anaesthesia. It carries with it the risk of explosion, depression of the vital centres and, in the case of chloroform, primary cardiac failure. With the introduction of the muscle-relaxants the practice has arisen of using light general anaesthesia in combination with one of these agents. Of this method Professor R. R. Macintosh[1] says: 'Some anaesthetists use light general anaesthesia and prevent reflex responses with curare (or its equivalent) but the dose is so large that respiratory arrest results. The patient is then inflated with oxygen and what might be termed intermittent bronchoscopy is carried out during periods of apnoea. This method involves some degree of haste and is not without risks.'

The method described below has proved its usefulness and value and its freedom from the disadvantages of methods previously used:

The usual premedication is pethidine, 100 mg., and atropine, gr. 1/100, for an adult.

On the operating table the patient is given pentothal sodium, the average dose being 0·25—0·3 g., and when unconscious succinylcholine ('scoline'), 50-75 mg., is administered according to the size of the patient. The lungs are inflated 4 or 5 times with oxygen from the anaesthetic machine.

A mechanical respirator is then placed in position and artificial respiration is carried out until the examination is completed. We use the Kifa respirator. This apparatus consists of two main parts, viz. (1) a set of four shells of different sizes, and (2) a power unit operated electrically. The shell and power unit are connected by an air line of flexible accordion rubber tubing. The metal shells cover the breast and abdomen of the patient and in no way interfere with the manipulations of the surgeon.

Further doses of pentothal sodium, to keep the patient unconscious and succinylcholine to produce apnoea are administered as required. Before introducing the bronchoscope the surgeon paints the cords with 2% xylocaine hydrochloride. As soon as the bronchoscope is introduced oxygen is passed down its side tube.

At the termination of the procedure the respirator is removed. The patient usually commences breathing within a very short interval. Until natural breathing recommences the patient is inflated with oxygen from the anaesthetic machine as and when necessary.

As the patient has been kept under light anaesthesia consciousness and reflexes return rapidly.

DISCUSSION

The technique described above provides conditions excellent for bronchoscopic examinations and biopsy. We have not yet had occasion to employ this technique for the removal of foreign bodies but there can be no objection to its use for that purpose*. The surgeon is unhurried and can be thorough in his examination. Toxic inhalation agents are not used. There is no risk of explosion. The patient is well oxygenated throughout the procedure, and the surgeon is not hampered in his work. By keeping the dose of pentothal low, respiratory depression at the end of the examination is obviated and we have not encountered any case of prolonged apnoea following succinylcholine.

I should like to record my indebtedness to Mr. A. E. Laubscher, Head of the Department of Surgery for constant encouragement in the development of this technique.

ADDENDUM

Since this paper was written we have had occasion to employ this technique for the removal of a foreign body from the right bronchus.

A non-European female aged 14 years was admitted to the Boksburg-Benoni Hospital with the history of having swallowed a safety-pin. She was X-rayed and the radiologist reported as follows: 'An open safety-pin with the point upwards is present in the right

* See addendum.

main bronchus and the lowest portion has entered the main bronchus to the right lower lobe.'

It was obvious from the position of the pin that its removal would be difficult and the surgeon would require a clear operating field without any interference.

The patient was premedicated with atropine gr. 1/100, at 4 p.m. On the operating table at 4·45 p.m. 0·2 g. of pentothal and 75 mg. of succinylcholine was administered. The lungs were inflated with oxygen from the anaesthetic machine and the Kifa respirator set in motion. At 4·50 p.m. the surgeon began and worked unhindered till the safety-pin was delivered at 4·59 p.m. During the course of the operation a further 0·2 g. of pentothal and 75 mg. of succinylcholine was administered.

In this case the patient was apnoeic for a period of practically 14 minutes. She was well oxygenated throughout. There was never any cause for worry or hurry. A few minutes after the operation the patient's reflexes had returned, and she had completely recovered from the anaesthetic shortly after returning to the ward. She was discharged a few days later.

I wish to express my thanks to Dr. L. Jacobson, E. N. T. Surgeon, and Dr. P. N. Swanepoel, Medical Superintendent, for permission to publish this case.

REFERENCE

1. Macintosh, R. R. (1954): Anaesthesia, **9**, 77.

IN MEMORIAM

HARRY ELLIS BRAWN, F.R.C.S.

Dr. Harry Ellis Brawn, who died at Port Elizabeth on 28 November 1954 at the age of 78 years, was born at Lichfield, England, and came to South Africa during the South African War as a doctor in the Royal Army Medical Corps. After the war he returned to England and became a Fellow of the Royal College of Surgeons, England. He subsequently returned to South Africa and practised in Uitenhage. In 1909 he married Miss Grace Cecelia Heugh of Uitenhage.

Dr. H. E. Brawn

During World War I Dr. Brawn served with the R.A.M.C. in Gallipoli and East Africa. He was mentioned in despatches and received a personal letter of thanks from Mr. Winston Churchill. After this war he practised in Klerksdorp but later returned to Uitenhage, where he practised until 1932. In World War II, despite his age, Dr. Brawn again answered his country's call; joining the S.A.M.C. he was appointed A.D.M.S. to the Eastern Province Command; he subsequently served with No. 4 General Hospital in Egypt. He was discharged in 1943 with the rank of Lieut.-Colonel.

Although a general practitioner, Dr. Brawn was always keenly interested in surgery and in particular in gynaecology and obstetrics. In 1934 he was appointed Honorary Gynaecologist and Obstetrician to the Provincial Hospital, Port Elizabeth, having previously returned to England to further his studies in this speciality. He initiated and developed the ante-natal services of Port Elizabeth and their present advanced state of development is largely due to his inspiration and efforts. In 1949 he retired from the active staff of the Provincial Hospital and was appointed Consultant in his old department.

A keen rugby player, he joined the Swifts Club, Uitenhage, in 1903, captaining the side from 1905 to 1907 and playing for Eastern Province in 1906. One of his fellow players and club members was the late Mr. A. J. 'Sport' Pienaar. He was made a life member of the Club in 1909, President 1923—1926 and a Patron 1927—1951.

Dr. Brawn took a keen interest in all matters of interest and importance to his profession. He was an active member of our Association; he seldom missed a meeting of the Cape Midlands Branch and for many years served on the Branch Council; he was also a past President of the Branch and a member of the Medical Advisory Committee of the Provincial Hospital and of the old Provincial Hospital Board.

The life of Harry Ellis Brawn exemplified the very best in Medicine and will never be forgotten by those who knew him. He was truly loved, trusted and respected not only by his patients but by all his colleagues.

He gave of his best at all times, wholeheartedly and unstintingly, never sparing himself for rich and poor alike; and what a wealth of knowledge and sound common-sense he brought to bear in the practice of the vocation to which he so rightly responded all his life!

He is survived by his wife, a son and two daughters, one of whom is a doctor.

One of the Officers who served under Dr. H. E. Brawn during the last war writes: Lieut.-Col. Brawn was in charge of the Surgical Division of the first South African General Hospital to be sent to Kenya and Egypt.

Although getting on in years Col. Brawn cheerfully submitted to all the discomforts and rigours of tent life in the trying climate found in the tropics and Mediterranean area. One example lingers in my mind: on winter mornings, when most of us spent as long as we could in our tents, 'Daddy' Brawn would be up at daybreak for his regular cold shower!

By the example he set us, he showed that he was primarily in the field to treat patients and he would not tolerate any red tape that stood in the way of this. It was a privilege and an honour to serve under him.

REGINALD EDGAR MEAKER, M.B., CH.B., D.P.H. (CAPE TOWN)

Dr. H. Nelson, Medical Officer of Health, Pretoria, writes: The passing of Dr. R. E. Meaker is not only a great loss to his family, and to the many friends to whom he endeared himself, but it is indeed a great loss to the Public Health Services of South Africa.

I have known Reg for many years, but first got to know him intimately in the early years of the last War, when we worked together in the Hygiene Section of the S.A.M.C. It did not take long to realize that in him we had a man with extensive knowledge of his work and a great deal of quiet keenness and enthusiasm.

Apart from his work, he was one of those likeable characters to whom one is instinctively drawn. He had a quiet quick sense of humour which hardly ever left him. He did whatever he had to do with a deep sense of loyalty and devotion. At the end of the war, I was very pleased when he applied to join the Pretoria Municipal Health Department, and it was a happy day for me to renew our friendship. We worked together for a few years, and throughout his stay with us he displayed the same keenness and loyalty, endearing himself to the members of the staff and his patients.

I knew, however, that he would not stay long in a junior position and was not surprised when he was appointed Medical Officer of Health of Vereeniging. I was pleased for his sake, but it was a sad day for me and for many members of the staff—we missed this

cheerful colourful personality in our midst, and we missed him in our work. From Vereeniging he went to Paarl to develop a difficult area, and to bring to it his sound public health administration and breadth of vision. He worked very hard and only stopped when he had a serious heart attack in September of this year.

This paragraph out of his last letter to me, which I was busy reading when I received the telephone call to tell me the sad news of his passing, shows his courage and how devoted he had been to his work: 'The Paarl Council have given me leave until 31 December 1954. Although I feel almost fit to start I've been advised to take all the leave I can. At present I feel fitter than I have for years. I've been overworking for years and this had to come.'

Reg Meaker was taken from us at a very early age, but he had already done much for the Public Health Service of South Africa in so short a time, and was held in such high esteem by his colleagues that he was made President of the Health Officials Congress of Southern Africa held at East London during September. Unfortunately he was taken ill just before the Congress, and his Presidential address was read for him by someone else.

We shall miss him but we shall always remember him with affection. Our sincerest sympathies go to his wife and his relations.

22 December 1954

Other tributes to the late Dr. Meaker appeared in the *Journal* (p. 1114) of 25 December 1954.

QUESTIONS ANSWERED

INH MEDICATION IN PULMONARY TUBERCULOSIS

Q. Is there any justification in the treatment of pulmonary tuberculosis (infectious cases) for prescribing only INH tablets, regardless of whether the patient is ambulant or not?

A. In the light of present experience it is still agreed by the majority of chest physicians that isoniazid should not be used alone in the treatment of pulmonary tuberculosis.

The reason is that with INH medication alone bacterial resistance develops in the majority of patients in a few months' time. Sputa remain persistently positive and radiological clearing is usually disappointing.

Although some of these isoniazid-resistant bacterial colonies fail to kill guinea-pigs and might possibly be considered avirulent, this should be disregarded at present in clinical practice, until more is known about it.

It is imperative always to use drug-combinations, e.g. INH in combination either with PAS or with bi-weekly injections of streptomycin, as these delay the emergence of resistance to either drug for long periods. These remarks apply, of course, equally to ambulant or non-ambulant patients.

P. L. Salinger

PASSING EVENTS : IN DIE VERBYGAAN

Union Department of Health Bulletin: Report for the 6 Days ended 15 December 1954.

Plague. Cape Province:

Two (2) Coloured cases and two (2) Coloured deaths at Kalkputs in the Williston District. Diagnosis confirmed by laboratory tests. All necessary precautionary measures taken.

No further cases of plague have been reported from the Kroonstad District (Orange Free State) since notification of 18 November, 1954. This area is now regarded as free from infection.

Smallpox: Nil.

Typhus Fever. Cape Province.

No further cases of typhus fever have been reported from Mount Fletcher, Qumbu and Mount Frere Districts since notifications of 21 October, 1954, 11 November, 1954, and 18 November, 1954, respectively. These areas are now regarded as free from infection.

Epidemic Diseases in Other Countries.

Plague: Nil.

Cholera in Chalna, Dacca (Pakistan); Calcutta (India).

Smallpox in Karachi (Pakistan); Bombay, Cochin, Delhi, Kanpur (India); Moulmein (Burma); Phnom-Penh (Cambodia); Phanthiet, Saigon-Cholon (Viet-Nam).

Typhus Fever: Nil.

* * *

The Academy of Psychosomatic Medicine held its first Annual Meeting at the Plaza Hotel, New York City, on 8—9, October 1954. Over one hundred Fellows and guests attended the two day session, devoted to 'The Psychosomatic Aspects of Surgery.' The second annual meeting will be held again at the Hotel Plaza, on 6, 7, and 8 October, 1955. The general subject decided upon is the Psychosomatic Aspects of Drug Administration.'

* * *

National Medical Aid Society. A short while ago this Medical Aid Society circularized doctors to the effect that owing to a speeding up of their accounting system, accounts would be settled within 10 days of being received in the office of the Society, unless held up for certain reasons set forth in the circular letter. Doctors were advised to enquire if prompt payment had not been made. Certain doctors evidently took the instruction too literally and telephoned in some cases even before an account had been submitted by the member concerned. In all cases investigated, however, no unnecessary delay had been proved.

The General Manager of the Society has asked us to make it known that every endeavour is made to handle claims promptly. Owing, however, to the large number of claims dealt with daily, and for the reasons already mentioned in the Society's circular to doctors, it is not always possible to adhere strictly to the time limit, but in all cases payment is made at the earliest possible opportunity.

* * *

Essay Award Contest. The Council on Undergraduate Medical Education of the American College of Chest Physicians offers 3 cash awards to be given annually for the best contribution, prepared by any undergraduate medical student studying for a degree in medicine, on any phase of the diagnosis and treatment of chest diseases (heart and/or lungs). The first prize will consist of a cash award of $250. Second prize will be $100 and third prize, $50. The three winners will also receive a certificate of merit.

The winning contributions will be selected by a committee of well-known chest specialists and will be announced at the 21st Annual Meeting of the American College of Chest Physicians, to be held in Atlantic City, New Jersey, June 2-5, 1955. All manuscripts become the property of the American College of Chest Physicians.

Applicants are requested to study the format of *Diseases of the Chest* as to length, form, and arrangement of illustrations to guide them in the preparation of the manuscript. The following conditions must be observed: Five copies of the manuscript typewritten in English (double spaced) should be submitted to the Executive Director, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois, U.S.A., not later than 10 April 1955. The only means of identification of the author shall be a motto or other device on the title page and a sealed envelope bearing the same motto on the outside enclosing the name and address of the author. A letter from the Dean or Chairman of the Department of Medicine of the medical school certifying that the author is a medical student studying for his degree in medicine.

BOOK REVIEWS : BOEKRESENSIES

ENDOCRINOLOGY

The Year Book of Endocrinology (1953-1954 *Series*). Edited by Gilbert S. Gordan, M.D., Ph.D. (Pp. 390, with 99 illustrations. $6·00.) Chicago: Year Book Publishers. 1954.

Contents: 1. The Pituitary Gland. 2. The Thyroid Gland. 3. The parathyroid Glands and Calcium Metabolism. 4. The Adrenal Medulla. 5. The Adrenal Cortex. 6. Intersexuality. 7. Female Reproductive System. 8. The Testes. 9. Carbohydrate Metabolism and Diabetes Mellitus. 10. Endocrine Treatment of Neoplastic Diseases. Miscellaneous.

This book very adequately abstracts articles of endocrinological importance published between January and December 1953. The editor writes an introductory page or two at the beginning of each chapter, drawing attention to one or more outstanding advances in the realm of the particular gland to be dealt with. Frequently, too, he makes pertinent comments at the end of an abstract.

Unfortunately this book will be of use only to those with a sound knowledge of endocrinology. It amounts to no more than a series of abstracts of papers published during the year. A series of critical articles correlating co-ordinating and assessing the work done in each sphere of endocrinology, would be of much greater value not only to the average practitioner, but even to those whose particular interest is endocrinology. One such attempt is made in the book in the form of an article by Gilbert W. Kleman, whom the editor invited to survey the advances in our knowledge of nor-adrenaline. Similar articles on other topics would greatly enhance the value of the Year Book.

However, for those who require a compilation of abstracts of the year's publications on endocrinology, it is an eminently satisfactory book. B.G.S.

LEUCOTOMY

Prefrontal Leucotomy and Related Operations: Anatomical Aspects of Success and Failure. By Alfred Meyer, M.D. (Bonn) Professor of Neuropathology and Elizabeth Beck, Research Assistant, Institute of Psychiatry. (Pp. 60, with 20 illustrations. 10s. 6d.) London: Edinburgh: Oliver and Boyd. 1954

The final place which operations on the frontal lobe will have in the treatment of mental disorder has yet to be decided. The authors of this short and valuable monograph do not attempt such an assessment. Instead they provide what at this stage of our knowledge is much more valuable, namely accurate and detailed facts. They set out scientifically the results obtained and the conclusions, whether of psychiatric, surgical, anatomical or physiological importance, which can be drawn from the detailed analysis of 102 cases in which the brains were eventually recovered for study. These 102 were selected from a larger number solely on the arbitrary criterion of at least 5 months of post-operative survival, on the grounds that any lesser period prevented a considered assessment of the clinical result of the operation.

The anatomical extent of each operation is defined in detail by an admirable and easily understood method. In addition the study of the degenerations following the section of fibre-tracts to and from the frontal cortical areas has permitted a more precise neuro-anatomical definition of the relationship of this part of the cortex to other regions of the brain. The connections of the frontal lobe with the thalamus and hypothalamus are detailed with a clarity, precision and economy of words which cannot be overpraised. In addition the concept of the 'visceral brain' (the cerebral counterpart of the autonomic nervous system) and the idea that the good results of frontal leucotomies are due to disturbance of these parts of the cerebrum are carefully discussed.

The clinical data and anatomical extent of the leucotomy are set out in tables, case by case, and classified into 4 groups for convenience, viz. (1) predominantly affective conditions, (2) paranoid and schizophrenic psychoses with onset mainly over the age of 35, (3) schizophrenia with onset under the age of 35, and (4) miscellaneous cases. The correlation of the results in each group with the extent of the operation is clearly demonstrated, in so far as it emerges at all. A striking observation is that too posterior a cut involving the head of the caudate nucleus and the subcallosal bundle frequently leads to 'delayed death' from marasmus and uraemia.

The presentation of this material in this particular way will undoubtedly be of great value to neuro-surgeons and in addition there is much to interest the anatomist. The reputation of the Maudsley Hospital cannot fail to be enhanced by the issue of this monograph.

E.N.K.

ALLERGY

Primer of Allergy. By Warren T. Vaughan, M.S., M.D. Fourth Edition. (Pp. 191, with illustrations. £1 16s. 3d.) St. Louis: The C. V. Mosby Company. 1954.

Contents: 1. Learn to Live with your Allergy. 2. Abracadabra. 3. The Allergic Constitution. 4. The State of Allergy. 5. The Enemy—Allergens. 6. Defence of the Realm—Methods of Combating Sensitization. 7. Liaison and Reconnaisaance—Taking Inventory. 8. Plan of Attack—The Therapeutic Program. 9. A War of Attrition—The Long Drag. 10. The Minute Men in the Front Line. 11. Victory or Defeat. 12. General Orders—Directions to the Patient.

According to Punch, an American woman was recently granted a divorce on the grounds that her husband insisted on bringing home ham, to which she was allergic. Had she read this little book her marriage might have been saved. Like the reviewer, she might have been surprised to learn that the body resembles a country traversed by a long canal, on which barges are continually plying and unloading parcels which are carried away by little red messenger-boys. The frontiers of this country are constantly being menaced by hordes of nefarious Allergens, against whom the Antibodies, armed with syringe-guns, wage incessant warfare. There are illustrations too. Dr. Vaughan intended his book to be read by allergic sufferers, and doctors should find it genuinely useful in giving the intelligent patient an authoritative if somewhat forbiddingly highly-coloured account of his disability and how to live with it. The only serious fault of the book is its price. P.B.

BIOCHEMICAL INVESTIGATIONS

Biochemical Investigations in Diagnosis and Treatment. By John D. N. Nabarro, M.D., M.R.C.P. (Pp. 299+ix. 25s.) London: H. K. Lewis & Co. Ltd. 1954.

Contents: 1. Disturbances of Water and Sodium Metabolism. 2. Acid-Base Equilibrium. 3. Potassium, Magnesium, Iron, Copper. 4. Calcium and Phosphorus Metabolism. 5. Disturbances of Protein and Nitrogen Metabolism. 6. Disturbances of Carbohydrate Metabolism. Diabetes Mellitus. 7. Disorders of Fat Metabolism. 8. Diseases of the Gastro-Intestinal Tract and Pancreas. 9. Diseases of the Liver and Biliary Tract. 10. Biochemical Examination of the Urine: Investigation of Renal Function. 11. The Cerebrospinal Fluid. 12. The Endocrine Glands. 13. The Vitamins. 14. Poisoning. Tables of Normal Values. Index of Investigations. Index of Conditions, Diseases and Treatment.

Biochemical investigations are becoming increasingly important in all branches of medicine. The newer advances in biochemistry are widely scattered throughout the numerous journals and this book is an attempt at collating them. It is written by a physician as a practical guide for hospital interns and registrars.

The index is sound and is divided into 2 parts, an index detailing the requirements for the various investigations, and an index of conditions, diseases etc. Much of the chemist's time in the laboratory is wasted by the unsatisfactory state of specimens sent for analysis, and the first part of the index does the chemist a valuable service.

The subject matter, although only a guide, is strikingly up-to-date and a valuable feature is the very sound selection of review articles at the end of each chapter.

This book can be recommended as a sound introduction to the use of biochemical methods in diagnosis and treatment.

A.A.K.

SCHAFER'S HISTOLOGY

Schafer's Essentials of Histology. Edited by H. M. Carleton, M.A., B.Sc., D.Phil. and R. D. Short, M.B., B.S., Ph.D. (Pp. 661+xi with illustrations. Sixteenth Edition. 35s. 0d.) London: Longmans, Green and Co. 1954.

Contents: 1. Use of the Microscope: Examination of Common Objects. 2. Blood. 3. Blood. 4. Blood-Corpuscles of Ovipara. 5. The Amoeboid Phenomena of Leucocytes. 6. Epithelia. 7. Epithelia. 8. Connective Tissues. 9. Connective Tissues. 10. Cartilage. 11. Bone. 12. Bone. 13. Muscle. 14. Nerve-Fibres.

15. Nerve-cells. 16. Modes of Termination of Nerve Fibres. 17. Structure of the Larger Blood-Vessels. 18. Blood-Vessels. 19. Lymphoid Organs. 20. Endocrine Glands. 21. The Integument. 22. The Heart. 23. Respiratory System. 24. Structure and Development of the Teeth. 25. Lip: Tongue and Gustatory Organs: Mucous Membrane of the Mouth: Pharynx: Oesophagus. 26. The Salivary Glands. 27. The Stomach. 28. The Small and Large Intestine. 29. The Gall-Bladder and Liver. 30. The Pancreas. 31. The Kidney, Ureter and Bladder. 32. The Male Generative Organs. 33. The Female Generative Organs. 34. Central Nervous System. 35. Central Nervous System. 36. Central Nervous System. 37. Central Nervous System. 38. Central Nervous System. 39. The Eye. 40. The Nose and Ear. Appendices. Index.

This book needs no introduction as it must have been used by many thousands of students during the 70-odd years since it was first published. A bibliographical note states that this new idition—the 16th × completes the 133-thousandth volume.

The authors have made certain changes which add to the usefulness of the book. A number of illustrations have been changed for increased clarity and the better-quality paper used has enhanced their reproduction. The practical class instructions have been removed from the body of the text, where they formerly appeared at the head of each chapter, and have been grouped together as an appendix. This will prove a useful innovation.

This edition is a worthy follower of the high standard set by the earlier issues of this classic work.

A.H.T.

EAR, NOSE AND THROAT

Modern Trends in Diseases of the Ear, Nose and Throat. Edited by Maxwell Ellis, M.D., M.S., F.R.C.S. (Pp. 471+ix, 140 figures.) London: Butterworth & Co. (Publishers) Limited. South African office: Butterworth & Co. (Africa) Limited. 1954.

Contents: 1. Functions of the Labyrinth. 2. Aural Vertigo. 3. Hearing Tests. 4. Hearing Aids. 5. Children with Defective Hearing. 6. The Surgery of Non-Suppurative Deafness. 7. The Non-Surgical Treatment of Deafness. 8. The Chemotherapy of Acute Aural Infections. 9. Otitis Externa. 10. Acute Otitis Media. 11. Chronic Otorrhoea. 12. Intracranial Complications of Aural Infections. 13. Facial Paralysis. 14. Nasal Catarrh. 15. Upper Respiratory Tract Infection in Children. 16. Orbital Complications of Sinus Infections. 17. Osteomyelitis and Intracranial Complications of Sinus Infections. 18. Tumours of the Nasopharynx. 19. Laryngoscopy and Bronchoscopy. 20. Tuberculosis of the Upper Air Passages. 21. Stenosis of the Larynx. 22. Laryngeal Paralysis. 23. Treatment of Cancer of the Larynx. 24. The Patient after Laryngectomy. 25. Pharyngeal Diverticulum. 26. Cancer of the Pharynx. 27. The Oesophagus. 28. Speech Therapy for Children with Cleft Palate. 29. Radiology of the Ear, Nose and Throat. Index.

A new addition to the excellent Modern Trend series, this volume has its chief value for the advanced post-graduate student and the practising specialist. In few branches of medicine have there been such marked changes of basic conceptions over the past 2 decades as in diseases of the ear, nose and throat.

The advent of antibiotics, appreciation of allergic mechanisms and pathology, advances in radiotherapeutic techniques, new conception of normal nasal physiology, and advances in the application of electronics, have profoundly altered the whole face of the speciality. There has been a marked shift from the purely surgical approach, and an increased emphasis on medical aspects. On the surgical side there have been great advances in technique, with the common use of whole blood and its fractions, and the over-all cover by antibiotics. All of these aspects are features of this volume.

This book is open to the general criticism of works by multiple contributors, namely that it lacks consistency in style and approach. This is more than compensated for by the quality of the contributors, all of whom enjoy world-wide repute. The chapters on surgical treatment of carcinoma of the pharynx and larynx are of outstanding interest.

Many of the chapters are of interest to workers in other fields. The chapters on aural vertigo, otitis media, nasal catarrh and the radiotherapeutic control of infection are excellent, and of wide general interest. The volume is well presented on good quality paper with clear diagrams and good illustrations, and is a very worthy addition to the series.

B.G.

ORTHOPAEDICS AND TRAUMATIC SURGERY

The Year Book of Orthopaedics and Traumatic Surgery (1953-1954 *Year Book Series*). Edited by Edward L. Compere, M.D., F.A.C.S., F.I.C.S. (Pp. 362, with 209 figures. $6.00). Chicago: The Year Book Publishers, Inc. 1954.

Contents: 1. Poliomyelitis. 2. Congenital Deformities. 3. Anatomy, Embryology, Pathology and Physiology of the Skeletal System. 4. The Epiphyses. 5. Osteomyelitis and Other Infections. 6. Tumors, Cysts and Fibrodysplasia. 7. Arthritis and Rheumatism. 8. Fractures and Dislocations. 9. The Spine and Pelvis. 10. The Neck, Shoulder and Arm. 11. The Hand and Wrist. 12. The Hip, Leg and Knee. 13. The Foot and Ankle. 14. Surgical and Diagnostic Technics. 15. Amputations and Prostheses. 16. Instruments, Appliances and Bone Banks. 17. Miscellaneous. Index.

This Year Book is again edited by E. L. Compere. Recent articles of interest to orthopaedic surgeons and allied specialities are summarized. If the reader should wish to consult the original articles, the references are available.

The editor's introductory remarks give a bird's eye-view of the year's literature. A welcome addition has been the terse editorial comments which follow some of the articles summarized. These remarks stimulate interest in the articles and are often invaluable to the reader. The reviewer is of the opinion that an increase in the number of comments would enhance the value of the Year Book.

Many excellent articles are summarized. The section on Tumors, Cysts and Fibrodysplasia contains some particularly good articles.

In the experimental field an article dealing with the effect of ultrasonic therapy on growing bone is abstracted. The authors noted a destructive effect. This sounds a warning note regarding the indiscriminate use of this type of therapy until more is known about the possible side-effects.

Further articles on the value of intra-articular injections of hydrocortisone are summarized. An interesting theory to rationalize the superiority of intra-articular hydrocortisone over cortisone injected locally into the joint cavities, is suggested.

The section dealing with Fractures and Dislocations includes several articles on intramedullary fixation. A paper demonstrating the use of an acrylic prosthesis for the fractured radial head is abstracted.

The chapter entitled the 'Hand and Wrist' includes many articles of great interest. This aspect of orthopaedic surgery is growing in importance and this section is well worth reading.

This little book will be an acquisition to the libraries of all orthopaedic and traumatic surgeons. It serves a useful purpose in keeping one abreast of recent developments.

G.D.

INDUSTRIAL MEDICINE AND HYGIENE

Industrial Medicine and Hygiene: Volume I. Edited by E. R. A. Merewether, C.B.E., O.St.J., M.D., F.R.C.P., D.I.H., F.R. S.Ed. Barrister-at-Law, with Foreword by The Lord Horder, G.C.V.O., M.D., F.R.C.P. (Pp. 426+ix.) London: Butterworth & Co. (Publishers) Limited. South African office: Butterworth & Co. (Africa) Limited, Durban. 1954.

Contents: 1. Work of the Factory Inspectorate. 2. Factory Legislation. 3. Statistical and Epidemiological Methods in Occupational Medicine. 4. The Organization and Operation of an Industrial Medical Department. 5. The Organization of Industrial and Occupational Nursing Services. 6. The Ministry of Supply Medical Service. 7. Medical Aspects of Coal Mining. 8. Medical Services in Inland Transport. 9. Medical Supervision in Shops, Warehouses and Offices. 10. The Young Worker. 11. The Disabled Worker. 12. Industrial Rehabilitation. 13. National Insurance Benefit for Sick and Injured. 14. Feeding the Worker. 15. Canteen Services in Industry.

When a book is launched under the editorship of Dr. E. R. A. Merewether, H.M. Senior Inspector of Factories and the foremost industrial physician in Britain today, an important event has taken place in medical literature. When the foreword by Lord Horder has been read and one becomes aware that the book is the first of 3 volumes on Industrial Medicine and Hygiene, it is at once recognised that this subject has arrived as a major speciality. That such is the case is made clear by the contributors, each a specialist in the field in which he writes.

The historical survey and wealth of detail (though not such as to obscure the wood) contained in each section but corroborates the truth of the statement that the history of industrial disease reflects some of the most important chapters in the progress of human civilization and social evolution.

Whilst each contributor writes with the familiar knowledge of the prevailing conditions in the United Kingdom, the basic principles apply to any country, but the chapters on 'The Factory Inspectorate', 'Factory Legislation', 'Medical Aspects of Coal-Mining' and 'Feeding the Worker' should be read with due regard

to the conditions obtaining in this country in relation to environment, climate, race and colour.

The chapter on 'Industrial Rehabilitation' is of more than topical interest in view of the recent 39th South African Medical Congress during which the accent remained on Rehabilitation, and also because of the publication by the British Medical Association of a monograph on 'The Rehabilitation and Resettlement of Disabled Persons', a summary of which appeared in the issue of this *Journal* of 25 September 1954 (p. 833).

The writer on the section devoted to 'The Organisation of Industrial and Occupational Nursing Services' makes the following important observation: 'A patient suffering damage at the hands of a nurse may be awarded damages against her. It is, therefore, essential that all members of the nursing staff shall be insured against such a claim.' Few organizations take steps to protect their nursing staff in this manner.

Every contributor rightly stresses the importance of team-work in connection with factory management, for little will be achieved without the mutual co-operation of the general manager, the consulting engineer, the chief chemist, the works manager, the welfare officer, the senior foreman and, of course, the medical officer.

This book bids fair to become the standard text-book on Occupational Medicine, and apart from the team mentioned in the above paragraph, it can be recommended to the recently-qualified casualty officer, who will find within its pages practical hints designed to ease his growing pains when dealing with industrial injuries referred to hospital; to the general practitioner (often also employed as part-time medical officer to one or more industries) whose practice may contain a comparatively large proportion of mine, factory and shop workers; and to the specialist physician who may find himself having to give an opinion on an industrial disease with a potential medico-legal setting.

To the full-time industrial medical officer in charge of the medical section of a large organisation this book is a "must".

J.B.L.

Industrial Medicine and Hygiene: Volume II. Edited by E. R. A. Merewether, C.B.E., M.D., F.R.C.P., D.I.H., F.R.S.Ed., Barrister-at-Law. (Pp. 541, with illustrations.) London: Butterworth & Co. (Publishers) Limited. South African Office: Butterworth & Co. (Africa) Limited, Durban. 1954.

Contents: 1. Occupational Skin Diseases. 2. Occupational Ophthalmology. 3. Occupational Psychology. 4. Medical Aspects of Compressed Air Illness. 5. Compressed Air in Civil Engineering. 6. Compressed Air Illness in Diving. 7. Precautions for men Working in Compressed Air. 8. Electrical Accidents. 9. Explosives. 10. Heating and Ventilation. 11. Lighting, Glare and Efficiency. 12. Intense Sound and Ultra-Sound. 13. Effects of Ionizing Radiations. 14. Protection Against Ionizing Radiations. Index to Volume Two.

This second volume of this work follows the same pattern as Volume 1. Of the 15 contributors in each volume 8 writers in Volume 1 are medically qualified as compared with 5 in Volume 2. This is so because a number of chapters, e.g. 'Precautions for Men Working in Compressed Air', 'Electric Accidents', 'Explosives', 'Heating and Ventilation', and 'Lighting, Glare and Efficiency', contain matter which lies more in the sphere of engineering, now inextricably woven into the texture of industrial hygiene for the better safety of workers in industry.

The opening chapter on 'Occupational Skin Diseases', however, is written by 2 medical contributors with international reputatison in their respective fields. One has become widely known for his masterly editorship of *Modern Trends in Dermatology;* the other, in her capacity as H.M. Deputy Senior Medical Inspector of Factories was one of the architects of the National Insurance (Industrial Injuries) Act of 1946, which in the U.K. has superseded the Workmen's Compensation Act of 1925.

Without his health and his ability to earn, a worker is bankrupt. A few short paragraphs in this section demonstrate how much nearer medicine is drawing towards the ideal in industry, which (theoretically) should be: No accidents, no injuries, no industrial diseases. Recently, in a judgment given in favour of an employee, the court decided that, 'Where an employer is making use of a dangerous process, it is not enough for him to have available somewhere in the factory the appliances necessary to minimize danger . . . He must see that the workers make use of the appliances provided, in the correct manner'. It is rightly pointed out that chilblains and urticaria may be classed as occupational diseases. Practical hints in patch-testing are given and cutaneous sensitizers are discussed in some detail.

The chapter on 'Occupational Ophthalmology' begins with a short description of the anatomy and physiology of the eye; later, in discussing safety measures in industry, one is made acutely aware of the axiom postulated by Sir Thomas Legge that, 'If you can bring some influence to bear external to the workman (that is, one over which he has no control) you will be successful in preventing industrial accidents', by the example of the shield fitted to a grindstone which will function only when the shield is in the correct position. An appendix clearly outlines the standard of vision necessary for the services, railways, post office, and police force.

The next chapter on 'Occupational Psychology' shows how the psychological departments established during the last war by the various services formed the basis on which the Medical Research Council founded its Unit for Research in Applied Psychology, and how welfare, group training, leadership and a knowledge of human relations heighten that broad incentive to work known as 'morale in industry', and how important has become the ecological approach in industry.

In the sections on 'Compressed Air Illness' a broad picture is given not only of the medical aspects but also the engineering principles employed in the interests of safety to the workmen.

The chapters on 'Heating and Ventilation' and 'Lighting, Glare and Efficiency' are all that can be desired in a book of this nature and in the section on 'Intense Sound' it is pleasing to find the assertion that uniform noise in a factory will have little effect on workers but where sound (noise) is produced by irregular and unperiodical vibrations, pathological changes may be expected eventually.

In no text-book on Occupational Medicine published in recent years can a chapter on Ionising Radiation be found which outlines this absorbing subject in so clear and informative a manner.

This book must certainly find its place beside Volume I of this series.

J.B.L.

CORRESPONDENCE : BRIEWERUBRIEK

SWIMMER'S ITCH IN SOUTH AFRICA

To the Editor: I am greatly interested in the article under the above title by Botha De Meillon and N. Stofberg published in your issue of 11 December 1954.

On p. 1062 we read in paragraphs 6 and 7: 'The area *where the infection occurred* is known to be free of human bilharziasis and our surveys have shown that none of the accepted vectors of *S. haematobium* or *S. mansoni* occurred there.' Pond snails, *Lymnaea natalensis* and *Bulinus tropicus* were collected and found to be shedding fork-tailed cercariae and with these some experiments were performed in mice and monkeys. None of them produced a dermatitis when applied to the skin of the monkeys. These experiments have not been concluded and it is hoped will be reported later when certain other data from the human volunteers become available.'

If, as is generally believed and presumably proved by research workers, the cercariae of the bilharzia parasite by piercing the skin can in certain subjects cause a rash and intense pruritus, can it be concluded that such is the only cause of Swimmer's Itch? Personally I do not incline to such a view. As a youth I was a victim of *S. haematobium*—one of many youths who contracted the disease in the Buffalo River which flows through King William's Town. We all fully understood the symptoms of the disease and

never on a single occasion did I see or hear of a rash or pruritus among those who bathed in pools of the river. In my own case, and presumably because I was far away from all possible sources of re-infection during my medical training in Scotland and England, the infestation gradually died out. I returned home in 1911 and, having engaged in general practice over a period of 34 years (1911-1945), I received among my patients numerous cases of bilharziasis among whom I do not remember ever having encountered one who complained of a rash with pruritus. I was therefore not impressed by statements bearing on 'itchy rash' as a physical sign of bilharziasis; like many others, I have held an open mind on that point.

In 1919, by way of a hobby, I launched out on a practical study of the microscopic flora and fauna of our ponds, rivers and streams, and more especially of dams and open vleis, in which I have discovered many interesting green algae including Nitella, the Charales and other Chlorophyceae. In order to recover unicellular algae, numerous protozoa, insect larvae, etc., I raked out masses of the plants referred to and with my bare hands squeezed out the water to be further examined. Almost invariably after doing this (at more than one shallow vlei) after several days a rash appeared between my fingers and along the ulnar sides of the palms of both hands. The rash consisted of small papules and vesicles and itching was intense. Washing with strong soap and hot water and daily application of Tar Dermament (Parke Davis & Co.) cured the condition within a few days. Naturally I blamed myself for having wrung out the pond plants with bare hands.

In one particular pond—rainwater in a disused quarry—about 20 Bantu children whose duty it was to herd oxen and sheep, bathed daily; I assembled them and ascertained that none suffered from haematuria. In this pond I had found *Physopsis africana*, *Lymnaea natalensis*, *Isidora tropica* and a species of Bullinus. The first of these contained cercariae with forked tails.

I did not feel justified in assuming that several eruptions of an itchy rash on my hands were cercarial in origin and I set out to search for a possible and more likely cause—plant irritation or otherwise. In the water from the ponds referred to I discovered numerous minute mites (Acarinae—Arachnoidea)—creatures whose heads and thoraces are fused into a cephalothorax. The bodies of some of these mites are brightly coloured—crimson, yellow, blue, green or brown. Their legs are many-jointed and beaded, having an appearance resembling the limbs of the well-known itch parasite *Sarcoptes scabiei*. I found numerous sharp spines on the limbs of these freshwater pond mites and came to the conclusion that these minute biting and suctoria parasites were, very probably, the cause of the rash which broke out on my hands. When the rash first appeared I am convinced that, by scratching, I conveyed the parasite or its eggs to both feet in the hollows beside the tendo Achillis and to which I also applied 'Tar Dermament'.

I am indebted to both writers of the valuable article referred to and while they are busy on their research experiments on Swimmer's Rash would respectfully invite them not to overlook the importance of the freshwater Acarinae as a possible cause of that distressful complaint. There would seem to be good reasons for regarding them as external skin parasites which fix themselves to the skins of bathers, and they are generally far more numerous than the cercariae of the bilharzia parasite. Their presence in ponds and vleis may account for rashes and pruritus contracted in the abscence of bilharzial cercariae, as I have learnt to my own cost.

A. W. Burton

39 Ayliff Street
King William's Town
C.P.
21 December 1954

TREATMENT OF DIARRHOEA

To the Editor: In response to my article on the treatment of diarrhoea in young children in general practice[1], Professor Findlay J. Ford[2] quite justifiably criticizes the absence of statistics, and he raises an eyebrow of scepticism at my statement that I have had no fatal case of gastro-enteritis over the past 3 years.

I have checked my records with those of the Registrar of Births and Deaths. Over the past 3 years I have certified the deaths of 28 children, and gastro-enteritis was given as the cause of death in only 3 instances.

In one case I was called to a farm where a Coloured family had become acutely ill after eating watermelon. They were rushed to hospital, the youngest member, aged 11 months, dying in the ambulance. The other 5 recovered in hospital.

In the second a Coloured infant was brought to my rooms in a critical condition after having been ill for 5 days. It died ½ hour after I had washed out the bowel and before any treatment instituted by me could be expected to have any effect.

In the third instance the death certificate was issued by me on behalf of a colleague who had left town and whose patient I had not attended during life.

The records further show that in 4 cases I gave the cause of death as either broncho-pneumonia or nutritional oedema with diarrhoea as a contributing factor. In my article I referred only to the common causes of diarrhoea as seen in general practice, no mention being made of the parenteral or symptomatic diarrhoeas, as the treatment outlined by me would not apply to these cases, or to the other less common causes of diarrhoea.

I think, therefore, that I am not making an overstatement when claiming that over the past 3 years I have not had any fatal case of gastro-enteritis in the sense implied by my article.

As the incidence of gastro-enteritis is highest amongst the non-European section of the community it would be impossible for any general practitioner to compile the accurate statistics which I agree would be highly desirable, and the findings would also vary according to the type of practice. Coloured patients constitute the cash element in any practice and are mostly seen by consultation. When a patient fails to return it is difficult for the doctor to know whether he is better or whether he has gone elsewhere. Any figures I give can only be regarded as approximate. Paarl has only 2 hospital cots for Coloured children. With the completion of our new hospital and the advent of an out-patient department it might be easier to follow up Coloured cases.

RECENT PRACTICE

My own practice is almost exclusively paediatric in character and about one-third of my patients are non-Europeans. The ratio of consultations to visits for Coloured patients is about 5 to 1, while in Europeans this is more or less reversed, no doubt owing to considerations of economy as opposed to convenience, for the main street is 9 miles long and Paarl is also largely a farming community.

I estimate that I have treated about 1,000 cases of diarrhoea in children over the past 3 years. This would not include the milder dietetic upsets in infants where only correction of the diet would be deemed necessary. In most of my patients I have commenced treatment early, and I estimate that in not more than 100 cases was there evidence of dehydration when first seen by me, and that there was emaciation or marasmus in about 200 more. By experience I am aware that in hospital practice these figures would be more or less reversed.

In my practice more than half of the cases seen by me would fall into the category of dietetic diarrhoea. In hospital practice the large majority would be classed as infective.

I realize that I am giving impressions and not statistics, but if any apology is due it would be for the conditions which make it impossible for the general practitioner to obtain anything more concrete than impressions based on his experience.

My article was an attempt to describe to the general practitioner a line of approach to the treatment of diarrhoea in young children which, from my own experience, gives better results than the present orthodox treatment. It is based on the conviction that diarrhoea is nature's own effort to expel an irritant from the bowel and that to institute constipating measures in opposition would be irrational. I do not disagree with the accepted opinion that an inflamed bowel must be rested, but I do maintain that in order to rest completely it must be efficaciously emptied of undigested food or bacterial toxins. I have found that the best way of attempting to empty the bowel is by means of the bowel wash-out as described in my article. The second best way is by the use of castor oil (see my reply to Dr. van Biljon in this issue). In the early and milder cases of diarrhoea castor oil does not appear to have any harmful effect, and is preferred to the bowel wash-out, which is cumbersome and unpleasant. I did not mention the dosage of castor oil; I use the customary dosage, which is about one teaspoonful per year of age in young children.

Post-mortem findings are slight and frequently absent in gastro-enteritis. The most constant finding is fatty degeneration of the

liver, which is believed to be due to absorption of toxins from the bowel.

PAST EXPERIENCE

My line of approach is more than an untested assumption. It has been slowly evolved from observations based on experience over several years. As a houseman in the paediatric department of Groote Schuur Hospital in 1944 my enthusiasm for my new calling was soon damped by the many cases of gastro-enteritis which ended fatally. The routine treatment was starvation, kaolin, sulphaguanidine and, when dehydration supervened, intravenous dextrose and saline. The 'drip' was a success, the kaolin was a success, yet only too often the patient died from toxemia. To me clearly the treatment left much to be desired. Soon I came across parenteral diarrhoea, the first being a case of pyelitis which was only diagnosed after the infant had been on sulphaguanidine for 2 days. Since then I have preferred to use a more universal sulphonamide in the treatment of diarrhoea.

In 1947 I was serving with the British Red Cross in occupied Germany, being in charge of a 100-bed hospital for children from the refugee camps. The average number of deaths from gastro-enteritis was about 8 per day. Conditions in the camps were insanitary but better than the hovels occupied by many of our non-Europeans. Food shortage was acute and malnutrition and starvation widespread. The parents were fairly intelligent and the hospital facilities provided for me by the Red Cross were first-rate.

At this time I began employing castor oil initially, resorting to kaolin only after 24 hours' starvation, and was rewarded by a quicker response to treatment. Also about the same time I read with interest a series of articles in the *British Medical Journal* on the biochemical changes in dehydration and as a result gave Hartman's solution by mouth during the period of starvation. Glucose was added in a strength not exceeding 1 teaspoonful to 4 ounces. During a period of shortage cane sugar was substituted with no appreciable deleterious effect. By late summer the deaths had fallen to an average of 3 per day. Seasonal incidence could not have played a large part in this reduction for in Europe the incidence is greatest in autumn, but one could of course speculate on many other factors. The impression I gained was that the death rate had definitely fallen in relation to the number of cases treated.

It was also in Germany that I first noticed that the largest number of cases of febrile convulsions in young children was caused by some indigestible substance, and the vigorous use of the enema syringe was introduced with good effect. It was found to be the quickest way of reducing the high temperature, and was diagnostic as well as therapeutic in revealing usually, unripe raspberries or cherry skins.

PRACTICE AMONGST THE COLOURED

On entering general practice 7 years ago I met with the problem of the Coloured child acutely ill in unsanitary surroundings for whom no hospital bed was available, and I began combining all the methods which experience had shown me might be beneficial.

I have not found the vigorous use of the enema syringe to be a hazardous project. In my rooms my own nurse employs it and on calls I have to carry out the operation myself. I have no idea how high the water goes up but frequent peristaltic movements are set up and often it is only after an interval that a visible result is obtained. I have been impressed by the number of times I have noticed sand, pips, skins, and orange-pulp mixed with kaolin in the washings. These things I can see with the naked eye. I cannot see bacterial toxins but I know they are there and their presence can have no effect other than harmful.

The intraperitoneal infusion is recommended only as the simplest effective method in general practice of administering parenteral fluid as an emergency measure. Where economic considerations and circumstances permit, the general practitioner is well aware of the less crude methods of administering fluids parentally, and the better solutions which can be obtained in vacolitres. He will also know about hyalase, and its price.

For intraperitoneal infusions I customarily add one teaspoonful of common salt per pint when the water is taken off the boil. On one occasion the only salt offered was coarse and formed a scum on top of the water. I preferred to do without and used water only, with no bad effect, though salt was given per mouth. About 200 c.c. may be given intraperitoneally without apparently causing discomfort.

I agree with Professor Ford that adequate rest is essential in any acute illness. A dehydrated semi-comatose child can be roused to take a few sips, after which it immediately sinks back into its moribund state. The instruction to the mother that sips must be given at 10-minute intervals may be regarded as an overstatement in order to impress on her the importance of giving fluids frequently. Even if she were to carry out the instructions to the letter I do not think it would disturb this type of child. Where there is no vomiting larger amounts can of course be given at longer intervals.

It has long been customary to add sugar or glucose to the water during the period of starvation. Some believe that it would help to protect the liver. Others allow it as it appears to satisfy the mother, who is loath to let her child starve. Barley water, carrot water, and weak black tea have the same reassuring effect on the mother and therein lies their main value.

In expounding one's impressions it is only human to be biased in one's judgment. I have tried hard not to be.

Frank Rousseau

Pastorielaan
Paarl
23 December 1954

1. Rousseau, F. (1954): S. Afr. Med. J., **28**, 1038 (4 December).
2. Ford, F. J. (1954): Ibid., **28**, 1088 (18 December).

Footnote. Dec. 27. I append this footnote in reply to the letter in this issue from Professor Davel and Dr. Pretorius, which I have had the opportunity of seeing before publication. I am in agreement with their views as far as modern hospital practice is concerned, but in general practice it would be impract'cable to try to apply modern hospital treatment.

By far the largest majority of cases of diarrhoea are treated by general practitioners, and in the past 10 years the non-European death rate has fallen from 300 to 227, i.e. no appreciable fall at all, despite important advances in the treatment.

It appears to me that the majority of diarrhoea cases in general practice are seen early and are of dietetic origin more often than infective, and I think that under these circumstances castor oil would give quicker and better results than kaolin.

I am being misinterpreted by Professor Davel when he quotes me as saying that 'sulphonamide plus a constipating agent was disastrous'. In my original article I had also added 'with disregard of the time-honoured treatment of puragation followed by starvation'. I was thinking mainly of several cases I had come across where a mixture of sulphonamide and kaolin had been given, but the mother not instructed on the most important principle in the treatment.

F.R.

(A) BEHANDELING VAN BUIKLOOP IN BABAS
(B) DIE VOORHUID IN PASGEBORE BABAS

Aan die Redakteur: Ek sal graag wil antwoord op die vrae gestel deur dr. P. J. van Biljon in sy brief in u tydskrif van 18 Desember.

(*a*) Hy vra of, met betrekking tot die behandeling van diarree in babas, die gebruik van kasterolie nie te prikkelend op 'n reeds geïnflammeerde dermslymvlies sou wees, en of dit nie beter sou wees om een van die sout-purgeermiddels soos magnesium-sulfaat, te gebruik nie.

My persoonlike opvatting is dat kasterolie met een of twee werkings die derm goed ledig en dat 'n neiging tot hardlywigheid daarop volg, en dit is die wat ek in diarree sou verlang. Ek was nog altyd huiwerig om magnesium-sulfaat te gebruik, eerstens omdat dit deur osmose water trek en dus die gevaar van wateronttrekking mag verhoog. In 'n kleinere dosis sal dit nie die derm gou en deeglik ledig nie, en in groter dosis sal die purgering langer aanhou dan in die geval met kasterolie. Tweedens, waar ek gewoonlik 'n sulfamiddel in die behandeling voorskryf, is ek bang dat magnesium-sulfaat weens sy swaelbestanddeel die moontlikheid van 'n sulfareaksie mag verhoog. maar ek het geen ondervinding hiervan nie.

In verband met sy tweede vraag stem ek saam met dr. van Biljon dat glukose meer gisting in die baba veroorsaak en ook meer onekonomies is dan rietsuiker, en persoonlik verkies ek gewone suiker bo glukose om dieselfde redes. As hy my artikel weer sal nagaan sal hy sien dat ek aan die moeder ‚suikerwater met 'n bietjie sout' aanbeveel! As die kind oud genoeg is laat ek selfs

enige koeldrank en jellie toe, en natuurlik ook die ou aanbeveling van flou swart tee met suiker.

(*b*) Graag wil ek ook saamstem met alles wat dr. van Biljon sê in verband met die voorhuid van pasgebore babetjies. Ons moet dit as normaal beskou dat die voorhuid in die eerste paar jaar aan die glans vaskleef. Ek vind dit ook maar al te dikwels dat 'n vroedvrou dit as een van haar pligte ag om die voorhuid te rek en terug te forseer en sodoende klein barsies in die natuurlike voutjies mag veroorsaak wat dan vassweer en mag lyk tot 'n egte fimose.

Werklike vernouing is baie seldsaam. Gairdner[1] het bevind dat van 54 babas wat na die hospitaal verwys is vir besnyding, net een 'n fimose gehad het. In die ander was dit net 'n vasklewing van die voorhuid. By die ouderdom van 3 jaar was daar 10% van seuns waarvan die voorhuid nie teruggetrek kon word nie, en in 200 onbesnede seuns tussen die ouderdomme van 5 tot 13 jaar het hy bevind dat in slegs 6% die voorhuid nie kon teruggetrek word nie en in 'n verdere 14% dit gedeeltelik teruggetrek kon word. In die meeste van hierdie gevalle kon die vasklewing verbreek word met minimum forsering. Waar hierdie manipulasie dan nie slaag nie en daar 'n werklike vernouing in 'n paar seuns skyn te wees, kom hy tot die gevolgtrekking dat net in hierdie paar gevalle besnyding wenslik is, en dat die voorhuid ten minste vir die eerste twee jaar alleen gelaat moet word.

Frank Rousseau

Pastorielaan
Paarl
22 Desember 1954

1. Gairdner, D. (1949): Brit. Med. J., **2,** 1433.

BEHANDELING VAN BUIKLOOP

Aan die Redakteur: Na aanleiding van dr. Rousseau se artikel[1] waar hy 'n paar opmerkings maak waarmee ons glad nie kan saamstem nie, en 'n metode van behandeling voorstel wat volgens ons mening selfs 'n skadelike invloed kan hê, wil ons graag die volgende kommentaar lewer:

Die roetine-gebruik van kaolin word as ‚gevaarlik' bestempel, en waar van die sulfonamide tesame met 'n stopmiddel toegedien word, was die resultaat ewe noodlottig'. Met enige dehidrasie word gewoonlik van die oplosbare sulfonamides gebruik gemaak en met of sonder 'n stopmiddel is nog geen rampspoedige of noodlottige gevolge waargeneem nie.

‚Na die dehidrasie herstel is, beswyk die kinders aan toksemie.' As oorsaak van dood speel ‚toksemie' sekerlik nie so 'n groot rol as die elektroliete-verlies en wanbalans nie, en die pasiëntjies wat ‚na die herstel' nog beswyk, is waarskynlik as gevolg van die ewe belangrike kaliumtekort[2]. Kalium word alhier as deel van die roetine-behandeling vir gastro-enteritis toegedien, dit is natuurlik per mond. Hier is die skrywer se behandeling met die intraperitoneale toediening van ‚gekookte' water, sekerlik 'n gevaar, afgesien van die tegniek om 'n gewone lumbaalpunksienaald (met 'n gemiddelde lengte van 4½ duim) tussen die derms in te druk. As geen elektroliete by hierdie water gevoeg word nie, en veral as die pasiënt nog opgooi, word die lewensbelangrike elektroliete so uit die liggaam uitgewas en 'n toestand van verminderde osmolêre konsentrasie verkry[3]. As 'n dehidrasie so erg met asidose of meer selde, alkalose is, dan moet die vogverlies en elektroliete-balans met die binne-aarse toediening daarvan herstel word. Hierdie is sekerlik nie altyd moontlik in 'n besige algemene praktyk nie; as tweede beste is die metode om die vog onderhuids met behulp van hyaluronidase toe te dien, sekerlik verkieslik bo intraperitoneale infusie. Die apparaat hiervoor kan oor en oor gebruik word en die enigste voorbehoud is nog 'n redelike mate van perifere sirkulasie en die toepassing van asepsis.

Die gebruik van kasterolie vir gastro-enteritis behoort by ons grootvaders gelaat te word. Dit veroorsaak net verhoogde peristaltiek met verergering van die kramppyne, verhoogde vog en elektroliete-verlies. Die publiek het al net begin afsien van die gewoonte om die ‚maag skoon te was' met kasterolie, veral na die skoolvakansies, en ook om dit vir enige koorstoestand of maagpyn te gee, en behoort nou daar gelaat te word. Om kasterolie te vervang met een van die soutpurgeermiddels soos magnesiumsulfaat, soos deur een van u korrespondente[4] voorgestel, sal die toestand nog meer vererger deurdat die purgerende werking van hierdie sout deur water-onttrekking is, met daarby elektroliete-verlies.

Oor die gebruik van suiker is alreeds kommentaar gelewer[4, 5]. Om egter glukose met rieksuiker te vervang[4], is nutteloos; rietsuiker, hoewel 'n disaccharied, gis nog net so maklik as glukose. Om gisting te vermy, moet liewer die alombekende dekstrimaltose gebruik word.

Weens die beweerde afwesigheid van enige mortaliteit van die skrywer se gevalle, moet ons met daardie behandeling aanneem dat sy pasiënte óf baie vroeë gevalle van gastro-enteritis was, lig aangetas óf uitermate taai.

Daar is nog vele ander punte waarmee nie saamgestem word nie, sommige is alreeds bespreek[5], en andere sal seker nog kommentaar uitlok.

Ten slotte onderskryf en stem ons saam met die opmerkings van prof. Ford[5].

J. G. A. Davel
H. P. J. Pretorius

Departement van Kindergeneeskunde
Universiteit van Pretoria
Pretoria
22 Desember 1954

1. Rousseau, F. (1954): S. Afr. T. Geneesk., **28,** 1038 (4 Desember).
2. Gamble, J. L. (1952): *Chemical Anatomy, Physiology and Pathology of Extracellular Fluid.* 5e ed., Chart 40 B. Cambridge, Mass.: Harvard University Press.
3. Moyer, C. A. (1952): *Fluid Balance,* 1e ed., p. 41 en p. 163. Chicago: Year Book Publishers Inc.
4. van Biljon, P. J. (1954): S. Afr. T. Geneesk., **28,** 1087 (18 Desember).
5. Ford, P. J. (1954): *Ibid.*, **28,** 1088.

DIE VOORHUID VAN PASGEBORE BABAS

Aan die Redakteur: Tereg maak dr. P. J. van Biljon[1], syns insiens grondig, beswaar teen menings wat in die Tydskrif ge-opper is en dit is op dieselfde grondslag dat ek met hom verskil oor sy bewering dat die huid nie vóór die ouderdom van 16-18 maande oor die glans kan gly nie omdat die praeputium tot op daardie ouderdom normaalweg met die glans verkleef is. Dit kleef wel vas maar dit kan maklik met behulp van 'n stomp wondyster tot by die *corona glandis* geskei word en om dit te bewys is dit nie nodig om die voorhuid terug te trek nie. Ek dink nie dat dr. van Biljon die moeders se verklarings dat vuilgoed onder die voorhuid saampak, bloot op grond dat die verklewing tussen die naasliggende huidlae dit onmoontlik maak in twyfel moet trek nie. Dit word tog algemeen ondervind dat wanneer die praeputium losgemaak en teruggedruk word, heelwat *smegma* agter die *corona glandis* gevind word. In baie jong babetjies (maar nie in pasgeborenes nie) is hierdie *smegma* geneig om hard te word en aan die *corona* vas te sit sodat 'n *focus* van kroniese irritasie ontstaan wat later tot etterafskeiding mag oorgaan.

Theodore James

Spring Gardens 16
Pinelands
Kaap
18 Desember 1954

1. van Biljon, P. J. (1954): S.-Afr. T. Geneesk., **28,** 1087 (18 Desember).

THE SIGNIFICANCE OF HAEMOPTYSIS

To the Editor: In our article under this title as published in the *Journal* of 4 December 1954 (page 1029) the first paragraph contains the words: 'The family practitioner with little to help him but the history and clinical examination, and often woefully deficient in the diagnosis of chest pathology, tells the patient that . . . ' It was far from our intention to describe the family practitioner as 'woefully deficient in the diagnosis of chest pathology'. The words in our MS as submitted were: 'The family practitioner with little to help him but the history and clinical examination (often woefully deficient in the diagnosis of chest pathology) tells the patient that . . . '

David Adler
Dennis Fuller

Johannesburg
22 December 1954